To Smoke or Not To Smoke:

Predictors of Smoking Behavior in People with

Head and Neck Cancer and Chronic Obstructive Pulmonary Disease

A Thesis

Submitted to the Faculty

of

Drexel University

by

Kim Phillips Baron

in partial fulfillment of the

requirements for the degree

of

Doctor of Philosophy

March 2003

DEDICATIONS

In Loving Memory

Of My Father

Lee W. Phillips

ACKNOWLEDGEMENTS

I wish to thank the many individuals who, in a variety of ways, contributed to the development and completion of this project.

I wish to thank the members of my dissertation committee for all of their invaluable efforts. First and foremost, I wish to thank Christine Maguth Nezu, Ph.D., A.B.P.P., chairperson of my dissertation committee. Dr. Nezu has provided me with great advice, feedback, and emotional support since the inception of this project. She also has been an exceptional clinical supervisor and mentor to me since my first year of graduate training and has taught me so much more than I ever expected to learn when I set out on this endeavor. Thank you to Pamela A. Geller, Ph.D., and Arthur M. Nezu, Ph.D., A.B.P.P., for their service on my dissertation committee as well as for their invaluable clinical supervision throughout my graduate training. I wish to thank Stephanie H. Felgoise, Ph.D., for her critical reviews of, and insights into, my work throughout the process, as well as her professional guidance, mentorship, and friendship. Finally, I would like to express my gratitude to Marylou K. McHugh, Ed.D., R.N., for her time and valuable recommendations for improvement.

I wish to thank the many doctors, nurses, and administrative staff at the sites where I collected data. I thank Dr. Edward Volkman, for introducing me to Dr. John Travaline, and an additional thanks to Drs. Daniel Kelly, Richard Hayden, William Sexauer, and John Travaline, for allowing me to work with their patients. Thanks to Tracy Costigan, Ph.D., for allowing me to seek her statistical consultation. Thanks to Candace Girdwood, for helping me to collect data while I was on internship. Thanks to

the many people who participated in this research project and took the time to talk with me and provide personal information.

Mostly, I wish to thank my family, for it is their day to day support and love that are most important to me. I thank my mother, Gala, for teaching me the importance of education, for encouraging me to pursue a career, for being proud of me, and for giving me her unconditional love. I thank my step-father, Stanley, for sitting down with me one summer day, asking me to think about what I enjoy, and giving me the confidence and support to turn that interest into a profession. I thank my brother, Rob, and his wife, Rachel, for their friendship and good humor. I thank my mother-in-law, Susan, for her encouragement and for watching Samantha many times so that I could complete my work. I thank my husband's grandmother, Bertie, for all her words of wisdom, and who has been like a grandmother to me. I also wish to acknowledge my father-in-law, Bob, his wife, Valerie, and my sister-in-law, Blaire.

Finally, I thank my husband, Rob, who has supported me during the challenges of graduate school, and has celebrated with me during the successful times. Thank you, Rob, for your love, your friendship, and your endurance in seeing me through this. Thank you also for making sure I took breaks. And finally, thank you, my sweet daughter Samantha: you make me smile each and every day, and remind me of what is really important in this world.

TABLE OF CONTENTS

LIST OF TABLES x

LIST OF FIGURES xii

ABSTRACT xiii

1. INTRODUCTION. 1

2. HEAD AND NECK CANCER 3

    Etiology and Epidemiology 3

    Diagnosis 9

    Treatment 11

    Psychosocial Adjustment 11

3. CHRONIC OBSTRUCTIVE PULMONARY DISEASE 13

    Etiology and Epidemiology 13

    Diagnosis 17

    Treatment 18

    Psychosocial Adjustment 20

4. VARIABLES ASSOCIATED WITH SMOKING BEHAVIOR IN THE GENERAL POPULATION 21

    Introduction 21

    Demographic Variables 22

        Age 22

        Gender 23

        Education 25

Income 26

Knowledge and Risk Perception 26

Smoking History Variables 28

Social Support 30

Depression 31

Anger 34

Coping and Problem Solving 35

Self-Efficacy 36

Locus of Control 37

Summary of Variables Related to Smoking Behavior in the General Population 38

5. VARIABLES ASSOCIATED WITH SMOKING BEHAVIOR IN THE CANCER POPULATION 40

Introduction 40

Demographic variables 40

Age 40

Gender 41

Education 41

Income 42

Ethnicity 42

Disease Severity and Treatment Variables 42

Smoking and Alcohol Variables 45

Psychosocial Variables 47

6. VARIABLES ASSOCIATED WITH SMOKING BEHAVIOR IN THE COPD POPULATION 49

Introduction 49

Demographic Variables 49

Age 49

Gender 50

Education 51

Income 51

Ethnicity 52

Disease Severity Variables 52

Smoking and Alcohol Variables 52

Depression 54

Self-Efficacy 54

7. MEASUREMENT OF SMOKING VARIABLES 56

8. SUMMARY OF SMOKING 62

9. RELEVANT THEORIES 63

Introduction 63

The Health Belief Model 63

Transtheoretical Model (Stages of Change) 68

Theory of Self-Efficacy 70

Social Problem Solving 72

10. THE PRESENT STUDY 82

Rationale 82

Original Research Questions and Hypotheses 85
Rationale for Revising Research Questions and Hypotheses 87
Revised Research Questions and Hypotheses 88
11. MATERIALS AND METHODS 91
Participants 91
Materials 94
Demographic and Smoking History Information 94
Social Problem-Solving Information 95
Mood Information 97
Patient Diagnostic Information 98
Procedure 98
12. ANALYTIC APPROACH 100
13. RESULTS 104
Descriptive Data According to Medical Diagnosis 104
Demographics 104
Medical Variables 107
Smoking Variables 111
Psychosocial Variables 116
Descriptive Data According to Smoking Status. 117
Demographic Variables 117
Medical Variables 117
Smoking Variables 118
Psychosocial Variables 125

Logistic Regression....126
Multicollinearity....131
Residual Analysis....131
Logistic Regression with Outlier Removed....132
Analysis of Response Operating Characteristics....137
Secondary Analysis....143
14. POWER ANALYSIS....145
15. DISCUSSION....147
16. LIMITATIONS....156
17. FUTURE RESEARCH....161
18. SUMMARY....166
LIST OF REFERENCES....167
APPENDIX A: Demographic Form....186
APPENDIX B: Social Problem-Solving Inventory - Revised....194
APPENDIX C: Profile of Mood States....195
APPENDIC D: Patient Diagnostic Form....196
APPENDIX E: Informed Consent MCP Hahnemann University....198
APPENDIX F: Informed Consent Temple University Hospital....202
APPENDIX G: Three-Month Follow-Up....206
VITA....210

LIST OF TABLES

1. TNM stage groupings for cancers of the head and neck. 10

2. Reasons for not participating. 93

3. Pearson correlations between predictor variables and outcome variable. 102

4. List of predictor variables examined.....103

5. Means, standard deviations, and medians for demographic data according to diagnosis 105

6. Frequencies and percents for demographic data according to diagnosis 106

7. Participants with head and neck cancer broken down by primary site 108

8. Participants with head and neck cancer broken down by TNM stage at diagnosis 109

9. Participants with COPD broken down by FEV1% predicted 110

10. Smoking status according to diagnosis 111

11. Means, standard deviations, and medians for smoking variables by diagnosis 113

12. Frequencies and percents of smoking variables according to diagnosis.....114

13. Means, standard deviations, and medians for psychosocial variables by diagnosis 116

14. Means, standard deviations, and medians for smoking history variables according to smoking status 119

15. Frequencies and percents for smoking history variables according to smoking status ...121

16. Means, standard deviations, and medians for psychosocial variables according to smoking status.....125

17. Variables entered in the stepwise logistic regression analysis for smoking status 128

18. Variables in the equation.....129

19. Classification table for model 130

20. Variables entered in the stepwise logistic regression analysis for smoking status with outlier removed 134

21. Variables in the equation.....135

22. Classification table for final model.....136

23. Coordinates of the ROC curve 141

24. Classification table for final model based on ROC analysis.....142

25. Frequencies of current smokers and former smokers in the head and neck cancer group according to TNM stage at initial diagnosis 144

26. Frequencies of current smokers and former smokers in the COPD group according to FEV1% 144

LIST OF FIGURES

1. Curve for response operating characteristics 139

ABSTRACT

To Smoke or Not to Smoke: Predictors of Smoking Behavior in People with Head and Neck Cancer and Chronic Obstructive Pulmonary Disease

Kim Phillips Baron

Christine Maguth Nezu, Ph.D.

Research demonstrates that smoking is highly correlated with, and a cause of, head and neck cancer and Chronic Obstructive Pulmonary Disease (COPD). Although the prevalence of smoking has declined since 1965, a significant proportion of the population continue to smoke. In addition to smoking being a cause of cancer and COPD, smoking has also been implicated in the progression of disease-related symptoms, diminished treatment outcome, and recurrence of disease. Previous research demonstrates that approximately 70% of individuals with COPD continue to smoke as compared to approximately 30% of individuals with head and neck cancer. Most of the studies have focused on demographic, smoking, disease severity and treatment variables, with little attention to psychosocial variables. Whereas demographic variables are stable variables which cannot be reversed, psychosocial variables are important to investigate not only because they may serve as obstacles to smoking cessation, but also, because interventions exist which would allow health professionals to intervene and increase smoking cessation success. This study investigated psychosocial predictors of smoking cessation in the head and neck cancer and COPD populations. One-hundred and three participants completed a semi-structured interview of their smoking history and two self-report questionnaires, the Profile of Mood States and the Social Problem-Solving Inventory - Revised. Fifty-four and 49 participants were diagnosed with COPD and with head and neck cancer,

respectively. Approximately 31% of individuals with COPD and 26% of individuals with head and neck cancer were current cigarette smokers, resulting in 29% of the combined sample currently smoking cigarettes. A logistic regression found that marital status, alcohol use, and depression predicted smoking status. The smoking history, problem-solving, and anger variables did not predict smoking status. In the final model, approximately 25% of the variance was accounted for. A response operating characteristics analysis yielded a model with 71% sensitivity, 68% specificity, and an overall classification rate of 70%. The results suggest that for individuals who are diagnosed with either head and neck cancer or COPD, and who report some distress, psychosocial treatment that addresses alcohol use and that is geared toward decreasing depression may help these individuals quit smoking.

CHAPTER 1: INTRODUCTION

Smoking accounts for 434,000 deaths annually in the United States (Cinciripini, Wetter, & McClure, 1998). In 1995, 24.7% of people in the United States considered themselves smokers, accounting for approximately 47 million adults (Centers for Disease Control [CDC], 1997). In 1996, 43% of men over the age of 18 and 33% of women over the age of 18 smoked cigarettes. Although the prevalence of smoking has declined by 40% between 1965 and 1990, this decrease reached a plateau between 1990 and 1995, and smoking prevalence has remained unchanged (National Center for Health Statistics, 1998).

Smoking is considered one of the few preventable behaviors and causes of morbidity and mortality (American Cancer Society [ACS], 1994; U.S. Department of Health and Human Services [USDHHS], 1990). However, although many smokers state that they would like to quit, only a small percentage are successful over the long term. Epidemiologic studies reported that in 1995, 45.8% of smokers had made at least one quit attempt during the past year, but only 23.3% were considered former smokers (CDC, 1997). In other words, approximately one half of the individuals who attempted to quit smoking subsequently relapsed. Similarly, in a community-level, 4-year smoking intervention study of over 20,000 smokers, smoking prevalence declined only 2.8% between 1988 and 1993 (Green, 1995b). These large scale findings show that many individuals continue to smoke cigarettes and that many who quit smoking have difficulty maintaining abstinence.

Smoking is strongly associated with many chronic diseases including cancer and pulmonary disease. Cigarette smoking is related to 30% of total cancer mortality and 82% of deaths from chronic obstructive pulmonary disease ([COPD], CDC, 1991). According to the CDC, the smoking-attributable mortality (SAM) in the year 1990 was 6,475 for individuals with cancers of the lip, oral cavity, and pharynx, 7,284 for individuals with cancer of the esophagus, and 2,990 for individuals with cancer of the larynx (CDC, 1993). Although the research shows a high correlation between smoking and cancer and smoking and COPD, many individuals with these illnesses continue to smoke. Research also shows that people who stop smoking, regardless of the age at which they quit, live longer longer (Taylor, Hassellblad, Henley, Thun, & Sloan, 2002).

The current study will investigate predictors of smoking behavior in people with either COPD or with head and neck cancer. The paper will begin with a brief overview of two illnesses that have been strongly linked to smoking behavior: head and neck cancer and COPD. Following this overview will be a description of current research on smoking behavior in three populations: the general population, head and neck cancer, and COPD. Several theories related to health behaviors such as smoking will be introduced. Particular attention will be paid to the problem-solving model. The current study will then be described, along with the results and a discussion.

CHAPTER 2: HEAD AND NECK CANCER

Etiology and Epidemiology

Head and neck cancer includes cancers of the lip, the oral cavity (i.e., the tongue, the inside lining of the cheeks, the floor of the mouth, the gums, and the hard palate), the paranasal sinuses and nasal cavity, the salivary glands (i.e., the parotid glands, the submaxillary or submandibular glands, the sublingual glands, the salivary glands, the pharynx (i.e., the oropharynx, nasopharynx, and hypopharynx), the larynx (i.e., the voice box, which includes the supraglottic larynx, the glottis, and the subglottis), the maxillary glands, and the thyroid gland. Together, head and neck cancers "account for 5 to 10 percent of all malignancies" (Helsper & Dollinger, 1991, p. 410). There are approximately 41,000 newly diagnosed cases of head and neck cancer per year (Moadel, Ostroff, & Schantz, 1998, p. 314). Approximately one third of people diagnosed with head and neck die of the illness (Helsper & Dollinger, 1991). The cure rate is considered "good" if the cancer is "found early, evaluated adequately and treated with the best available therapy" (Helsper & Dollinger, p.410). Five year survival rate, which is the most common statistic used to measure prognosis, is approximately 52% (Moadel et al., 1998).

Head and neck cancer has been strongly correlated to environmental pathogens. Tobacco use and alcohol use are considered to be the greatest risk factors for head and neck cancer. "Smoking contributes to 90% of all cancers occurring in organs that come in

direct contact with smoke, including the mouth, esophagus, lungs, and bronchus" (Hecht et al., 1994, p. 1658). Smoking is responsible for 87% of lung cancers as well as 30% of all cancer deaths. The American Cancer Society conducted two prospective studies on cancer prevention (Thun, Day-Lally, & Myers, 1995) and found that there has been a rise in cancer mortality due to smoking, and that smokers are more likely to die from cancer than non-smokers.

The relative risk of developing head and neck cancer is higher for current smokers than for former smokers. Relative Risk is defined as "the ratio of the incidence of those who are exposed to a risk (such as smoking two packs per day) divided by the incidence of the same disease in those who do not possess the characteristics (e.g., nonsmokers)" (Davis & Zitsch, 1999). Research has established that smoking increases the odds ratio of being diagnosed with cancers of the oral cavity and pharynx (Blot, McLaughlin, Winn, Austin, Greenberg, et al., 1988; Bungaard, Wildt, Fryenberg, Elbrand, & Nielsen, 1995; Talamini, La Vecchia, Levi, Conti, Farero, & Franceschi, 1998) and of the esophagus (Castellsague, 1999; Castellsague, Munoz, De Stefani, Victoria, Castelletto, Rolon, et al., 1999). Furthermore, individuals who quit smoking decrease their relative risk of developing cancer (Castellsague et al., 1999) as well as from dying of cancer (Hinds, Yang, Stemmerman, Lee, & Kolonel, 1982; Johnston & Ballantyne, 1977), whereas individuals who continue to smoke after their diagnosis increase their chances of developing another cancer (Newcomb & Carbone, 1992).

As examples to highlight the above points, one study found that smoking increased the risk of acquiring head and neck cancer 5.8 times (Stevens, Gardner, Parkin, & Johnson, 1983). A more recent study found that "male smokers have an 8.8-fold increased risk of developing cancer of the oral cavity" (Newcomb & Carbone, 1992), which is greater than never smokers, but less than current male smokers. Another study found that male smokers have a 22.5-fold increased risk of developing cancer of the oral cavity as compared to men who never smoked. In other words, men who currently smoke are approximately 22 times more likely to develop cancer of the oral cavity than men who never smoked. A recent study found tobacco and alcohol to be the strongest individual risk indicators and contributed significantly to the risk of developing oral cancer in 161 inpatients with intra-oral squamous cell carcinoma (Bundgaard et al., 1995). In a sample of 223 women with primary lung cancer, the risk of dying was significantly greater among ever-smokers than never-smokers (Hinds et al., 1982). In a sample of 351 patients with oral tongue cancer, 31% of the patients who had used tobacco, alcohol, or both were dead from tumors in five years, as compared to 14% of nonusers (Johnston & Ballantyne, 1977). In a sample of 115 patients with head and neck cancer, there was a significant difference in survival rates such that "the two-year survival rate was 66 percent in the group that did not smoke, as compared with 39 percent in the group that smoked" (Brownman et al., 1993, p.160).

Unfortunately, although smoking prevalence in the cancer population has declined, a significant number of individuals diagnosed with cancer continue to smoke. For instance, in a sample of 5,998 cancer patients with mixed diagnoses, current smokers and recent smokers

(defined as having abstained less than one year), smoking prevalence was 30% for males and 29% for females (Spitz, Fueger, Erikson, & Newell, 1990) and 18% of 688 males and females (Gritz, Kristeller & Burns, 1993). Samples consisting solely of smoking-related malignancies contain a larger percentage of current and former smokers. For instance, in two lung cancer studies, 51% of the sample were current smokers (Gail et al., 1984; Johnston-Early et al., 1980). More recent studies, which may reflect the anti-tobacco campaigns of the 1990s, have found smoking prevalence among head and neck cancer patients to be 62.5% at initial interview and quit rates to be 70.6% among males and 61.1% among females (Spitz, Fueguer, Chamberlain, Goepfert, & Newell, 1990). Sarna (1995) found that in a sample of 65 newly diagnosed lung cancer patients, five (7.7 %) were smokers during the time of the study, 51 (78.5 %) were former smokers, and 9 (13.8%) had never smoked. Of those who were former smokers, 26 (39.9%) quit in response to lung cancer, 15 (23%) quit at diagnosis, and 11 (16.9%) quit at surgery. Among a group of 840 lung cancer patients, 16.8% were smokers at one year follow-up and at 2 year follow-up, the cessation rate was at 40% (Gritz, Nisenbaum, Alashoff, & Holmes, 1991). In a sample of 144 patients with newly diagnosed squamous cell carcinoma of the upper aerodigestive tract, 35% of the 74 patients who had smoked the year before diagnosis reported continuing to smoke after surgery (Ostroff et al., 1995). In a review of smoking cessation among hospitalized patients with lung and head and neck cancers, “long term unassisted cessation rates … have ranged from 40% - 70%,” (Orleans, Kristeller, & Gritz, 1993. p. 784) revealing both the success of

smoking cessation programs as well as the difficulty of many patients in maintaining abstinence.

There are a variety of pathophysiologic mechanisms through which cigarette smoke causes cancer. According to Carbone (1992), tobacco causes mutations in tumor suppresssor genes (that ordinarily control cell growth) and dominant oncogenes (that cause unregulated cell growth when over-expressed). In addition, smoking causes "impaired mucociliary clearance" in the lungs and "decreased immunologic responsiveness" (Carbone, 1992, p. 15S) which can predispose an individual to cancer. In a group of 129 patients with squamous-cell carcinoma of the head and neck, "mutations of the [p53] gene correlated strongly with cigarette smoking" (Brennan, Boyle, Koch, Goodman, Hruban, Eby, et al., 1995, p.715).

As highlighted earlier, quitting smoking once diagnosed with cancer has important implications for survival. One possible explanation for the increased mortality among smokers in this population is that smoking decreases successful recovery of individuals already diagnosed with a chronic medical conditions by increasing complications and decreasing treatment efficacy. For instance, individuals who smoke during radiotherapy experience lower response rates and poorer survival as compared to individuals who discontinue smoking (Browman et al., 1993; Wynder, Dodo, Blooch, Gantt, & Moore, 1969). Several studies have found that continued smoking increases the risk of developing a second primary malignancy in the oropharygeal area (Day et al., 1994; Silverman, Greenspan, Grosky, 1983; Wynder et al., 1969). For instance, in a sample of over 1,000

patients with oral and pharyngeal cancer, tobacco significantly contributed to the risk of developing a second cancer (Day et al., 1994). The odds ratios rose with duration and intensity of smoking such that current smokers had a higher risk of developing a second cancer than former smokers (Day et al., 1994). This risk decreased for those who quit smoking at the time of diagnosis. Specifically, individuals who smoked 40 or more cigarettes per day had more than a threefold increased risk of developing a second cancer (Day et al., 1994). In a sample of over 2,000 men, the risk of esophageal cancer was found to decrease "rapidly, strongly and significantly with longer periods of abstention" (Castellsague et al., 2000). Likewise, Moore (1971) found that among 203 smokers considered cured of cancers of the oral cavity, larynx or pharynx, 40% of those who continued to smoke developed second cancers in "tobacco-contact tissues" versus only 6% of the nonsmokers (p.553). Stevens et al. (1983) found, in 269 patients with head and neck neoplasms, that those who continued to smoke after a diagnosis of cancer had a fourfold increase in the risk of recurrence over those who did not smoke, and twice the risk of recurrence over those who quit smoking. Moreover, smoking cessation and quitting alcohol reduces the risk of esophageal cancer after ten or more years (Bosetti et al., 2000).

Diagnosis

There is no routine screening for head and neck cancer. Often times, a dentist identifies a suspicious lesion and refers the patient to an otolaryngologist (a head and neck specialist). Symptoms of head and neck cancer vary according to the site of the cancer. Common signs include, but are not limited to, a "swelling or ulcer that does not heal ….sinus trouble that does not respond to antibiotics …. painless swelling and later paralysis of one side of the face …. difficulty or pain on swallowing …. enlarged lymph nodes in the neck …. and persistent hoarseness" (Helsper & Dollinger, 1991, p. 412). A diagnosis will include inspection of the oral and nasal cavities using mirrors and scopes, palpation of suspicious lesions, testing for the Epstein-barr antibody, x-rays of the sinus and skull, CT and MRI scans, and biopsies of suspicious lesions.

Cancers, including head and neck cancers, are typically codified for curability according to the TNM system whereby T represent tumors, N represents nodal involvement and M represents metastasis. Pathological staging is utilized to characterize not only the existence of a malignancy, but the extent to which tissues have been invaded by cancerous cells (Holland, 1998). This information is then combined, resulting in a severity rating ranging from Stage I (least invasive) to Stage IV (most invasive, includes metastasis to other areas of the body). (see Table 1). "Accuracy of staging is especially critical, since a slight difference in the location and size of the tumor has a significant effect on the therapy chosen, the extent of surgery and the prognosis (Helsper & Dollinger, p. 413).

Table 1. TNM stage groupings for cancers of the head and neck

Tumor	Nodal Involvement			
	N_0	N_1	N_2	N_3
T_1	I	III	IV	IV
T_2	II	III	IV	IV
T_3	III	III	IV	IV
T_4	IV	IV	IV	IV

Note. In addition, any tumor that has metastasized is classified Stage IV.

Treatment

Treatment for head and neck cancer depends upon the severity of the illness, and most frequently includes surgery, radiotherapy, chemotherapy, or a combination of these treatments. Therefore, individuals who are diagnosed at a later stage of illness are more likely to receive more invasive surgery, and are more likely to have post-operative radiation treatment. In addition to patients having surgery to remove the tumor, a majority of the patients require orthopedic reconstruction of the face and jaw.

Psychosocial Adjustment

Treatment for head and neck cancer often involves the aerodigestive tract as well as deconstruction of the face and jaw. Therefore, many patients experience not only facial disfigurement, but also exhibit difficulty swallowing, poor saliva production, and pain. In addition, other senses such as sight, hearing, taste, and smell are affected (Helsper & Dollinger, 1991). Patients may experience dry mouth, sensitivity to heat and cold, hoarseness, nasal discharge, and persistent drooling. Those with more disfigurement are at greater risk for psychological and interpersonal difficulties. Someone who experiences major disfigurement is also more likely to experience low self body-image and low self-esteem. Individuals can experience a range of psychological and emotional responses to their diagnosis and treatment including, but not limited to, shock, depression, anxiety, anger, and

fear. These can all lead to social withdrawal (e.g., from activities such as dining with friends or attending events). Although an instrument called a Servox now allows individuals whose voice box has been removed to verbally communicate, many patients are self conscious and hesitant to use the Servox in public.

CHAPTER 3: CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Etiology and Epidemiology

Fourteen million persons in the United States suffer from Chronic Obstructive Pulmonary Disease (COPD, American Thoracic Society [ATS], 1995). In 1991 there were 85,544 deaths due to COPD (ATS, 1995). An estimated 16.0 million Americans suffered from COPD in 1994, and this represented an increase in 60% since 1982 (National Center for Health Statistics, 1998). In 1995, COPD "ranked fourth among leading causes of death" (National Center for Health Statistics, 1998). There was a 40% increase in mortality due to chronic bronchitis and emphysema between 1979 and 1995 (National Center for Health Statistics, 1998).

Chronic obstructive pulmonary disease is "a disease of the airways characterized by airflow obstruction due to chronic bronchitis or emphysema" (ATS, 1995, p. S78). The purpose of the respiratory system is "to supply oxygen to the cells and to remove carbon dioxide" (Labott, 1998, p. 102). COPD has an insidious onset, is progressive in nature, and is primarily irreversible. Both emphysema and chronic bronchitis are associated with heavy cigarette consumption. Symptoms of COPD include dyspnea (shortness of breath), cough, sputum production, airflow limitation, impaired gas exchange, and frequent respiratory infections (Labott, 1998). Since there is no cure for COPD, treatment is both palliative and to reduce the risk of infection. While the clinical manifestations of chronic bronchitis and

emphysema are similar (i.e., both share the common symptoms of difficulty exhaling air from the lungs), and while the two share some of the same etiology (i.e., smoking and genetic risk factors), there are also some pathological differences. Chronic bronchitis is defined by its clinical manifestation, and is diagnosed with "the presence of chronic productive cough for 3 months in each of two consecutive years in a patient in whom other causes … have been excluded" (ATS, 1995, S78). Emphysema is defined according to anatomical pathology when there is "abnormal permanent enlargement of the air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis" (ATS, 1995, p. S78).

Tobacco smoke is, without doubt, a major risk factor for COPD. According to the ATS, the "primary cause of COPD is without question exposure to tobacco smoke" (ATS, 1995, S78). "Tobacco smoking accounts for an estimated 80 to 90% of the risk of developing COPD (U.S. D.H.H.S., 1984). Several studies conducted by Pederson and colleagues have found long-term cessation rates to average approximately 20% (Pederson, Baskerville, & Wanklin, 1982; Pederson Wanklin, & Lefcoe, 1991). The most recent large scale study of smoking cessation among individuals with chronic obstructive pulmonary disease followed 5,887 adult smokers with early COPD for 5 years (Murray et al., 1998). At baseline, 818 (20.1%) were sustained quitters, 1146 (20.1%) intermittent quitters, and 3711 (65.3%) continued smokers.

In a review of the pathophysiologic damage due to cigarette smoking, McKusker (1992) and colleagues cited a variety of ways in which the respiratory system is damaged.

More specifically, "smoke-induced damage to the lower respiratory tract may occur directly by oxidants carried within smoke particles …. By secretion or reactive oxygen species or degradative proteases …. By rendering inhibitors of enzyme activators inactive, or by changing the responsiveness of inflammatory cells to stimulatory events (See Sethi & Rochester, 2000, for review). Individuals who smoke show a greater annual decline in Forced Expiratory Volume (FEV1), a primary measurement of pulmonary function. (ATS, 1995).

Smoking cessation is considered the most important treatment for COPD (Labott, 1998), and improves prognosis regardless of age (Postma & Sluiter, 1989). Although quitting smoking neither reverses disease nor helps to regain significant lung function, individuals who quit may show an increased bronchodilator response (i.e., can breath more easily with the aid of inhalers), and in addition, a decreased rate of decline of lung function, becoming commensurate with individuals of the same age who never smoked (Anthonisen et al., 1994). In other words, research shows that quitting smoking after a diagnosis of COPD "has a beneficial effect on the course of COPD (Wise, 1997). For instance, among individuals with COPD, the decline in FEV1 (a measurement of expired air) is steeper for smokers than nonsmokers, and the heavier the smoker, the steeper the rate (ATS, 1995; Xu, Dochery, Ware, Speizer, & Ferris, 1992). COPD patients who continue to smoke show a decline in FEV1 that is twice the decline of those who quit smoking (Anthonisen et al., 1994). Unfortunately, continuous abstinence is low among COPD. Cessation rates during follow up periods ranging from 6 months to 7 years have been as low as 27%

(Pederson, Williams, & Lefcoe, 1980). Cigarette smokers have higher death rates from chronic bronchitis and emphysema and a higher rate of long term abnormalities (CDC, 1993). These differences increase in proportion to the quantity of smoking such that age of starting smoking, total pack-years and current smoking status are all "predictive of COPD mortality." (ATS, 1995, S79). Mortality rates for COPD are higher in smokers than in nonsmokers (ATS, 1995).

Interestingly, research has shown an "increase in COPD mortality even when the analysis was confined to active smokers (Wise, Szklo, Matanoski, & Neugut, 1997). Anthonisen (1994) and colleagues investigated the efficacy of a smoking cessation program plus inhaled bronchodilator therapy with individuals with mild obstructive pulmonary disease. Although the authors state that "the rates of 5-year sustained smoking cessation in these groups were among the highest ever reported for a major study" (p.1502), their 5-year cessation rate was only approximately 22%. Although the percentage of sustained nonsmokers was significantly greater than smokers in the usual care group (5%), this cessation rate is quit low. Smoking did not decrease significantly in either the intervention or control group of the Community Intervention Trial for Smoking Cessation ([COMMIT]; Green, 1995b), a prospective program which provided community intervention to reduce smoking.

Diagnosis

A diagnosis of COPD includes a detailed medical history, a smoking history, a physical exam, chest x-rays, blood tests and arterial blood gases, sputum culture and pulmonary function tests (PFTs; ATS, 1995). The most objective measurement available to make a differential diagnosis of COPD and to determine severity of illness is by administering a PFT. During spirometry (a component of the PFT), the patient is asked to breath in as much air as possible (in order to maximally fill the lungs) and then to exhale the air into a tube as quickly and as forcefully as possible, until he or she can no longer blow out any air. This procedure is often repeated three times for reliability. The instruments and methods for performing these tests are standardized by the American Thoracic Society. Measurements of spirometry include measurements of Forced Vital Capacity (FVC), which measures the total volume of air the patient expires after full inspiration, Forced Expiratory Volume in the first second (FEV1), which measures the volume of air the patient forcefully expires in the first second of exhalation, and the ratio of FEV1 to FVC. The "most useful indicator of disease severity, across all stages of disease, is spirometry, with FEV1 being the best indicator of abnormality" (Petty, 1993, p. 88). Forced expiratory volume is also "considered to be the best predictor of remaining life expectancy for [COPD] patients (Kaplan, Reis, Prewitt, & Eakin, 1994) and is the "best, most easily followed indicator of airway resistance in COPD" (Sethi & Rochester, 2000, p. 76). Among individuals with COPD, FEV1 is reduced as

compared to individuals with normal lung function. Disease severity measured by FEV1% takes into account individual differences such as size, gender, and age (Leidy & Traver, 1995, p.539). Outcome measures include the patient's actual flow rates as well as the flow rate as a percentage predicted for the patient based on normative data. In addition, FEV1 is considered a valid measure of airways obstruction due to its correlation with pathologic scores of airway diameter" (Enright, Lebowitz, & Cockroft, 1994, p. S10). Individuals with COPD display decreased flow rates as evidenced by a decrease in FEV1 and a decrease in the FEV1/FVC ratio. Airway obstruction has been defined as a ratio of FEV1 to FVC of 70% or less (Anthonisen et al., 1994). A diagnosis of COPD must include an FEV1/FVC less than 70% and a post-bronchodilator response of less than 80%. Although there is no current staging system, the ATS recommends a standardized categorization in order to "facilitate [the] approach to COPD ….[for] epidemiologic and clinical studies, health resource planning, prognostication, and the application of clinical recommendations" (ATS, 1995). They propose the following stages: FEV1% predicted >= 50% (and less than 70% predicted) stage I; 35-49%, stage II, <35% stage III.

Treatment

There is no cure for COPD. It is a chronic disease whereby treatment is designed to slow the progression of symptoms and severity. There are a variety of treatment options available to help patients manage their illness, slow down disease progression, reduce

symptoms, increase daily functioning, and improve quality of life. Such medical management may include smoking cessation; pharmacological interventions such as nicotine gum, the nicotine patch, or bupropion (e.g., Zyban®), to reduce the withdrawal symptoms of smoking cessation; bronchodilator therapy to "induce bronchodilation, decrease the inflammatory response, and facilitate expectoration" (ATS, 1995, p. S85); anti-inflammatory therapy (e.g., steroidal medications); psychotropic medications to treat depression, anxiety, insomnia or pain; oxygen therapy to reduce the risk of cellular hypoxia in individuals with hypoxemia; and pulmonary rehabilitation in order to "decrease respiratory symptoms and improve quality of life" (ATS, 1995, p. S93) by increasing exercise tolerance and thereby allowing patients to better engage in activities of daily living. Pulmonary rehabilitation includes education, exercise training, psychosocial intervention and support, and breathing retraining. Smoking cessation is an integral component to the treatment of COPD. Unfortunately, many individuals with COPD continue to smoke. For instance, "[c]ontinuous abstinence … may be as low as 27% in follow up periods ranging from 6 months to 7 years" (ATS, 1995, p. S84). Individuals with COPD who quit smoking may halt the decline, or loss, of lung function (See ATS, 1995, for review; Murray et al., 1998). However, over the long term, there is not a significant improvement in lung function (Anthonisen et al., 1994; Wise, 1997), such that the damage to the lung up to the point of quitting is relatively irreversible.

Psychosocial Adjustment

There are a variety of psychosocial sequelae including depression, anxiety, difficulty with relationships, sexual problems, and losses due to illness (Labott, 1998). Individuals with COPD experience a multitude of losses and health consequences such as "fatigue, dyspnea, and reduced exercise tolerance" (Dudley & Sitzman, 1988, p. 196). They suffer from a decrease and change in recreational activities, become increasingly dependent on others to assist in everyday activities, leading to changes in social roles. They often report a loss in positive body image subsequent to weight gain and use of home oxygen. Depression is common in the chronically ill, including individuals with COPD. Subsequent to changes in social roles and body image, relationships, including sexual relationships, often suffer. Many individuals with COPD report symptoms of depression. They also report fears, such as fear of breathlessness and fears of behaviors that can induce dyspnea (Greenerg, Ryan, & Bourlier, 1985). These fears subsequently lead to social avoidance and withdrawal, further intensifying feels of depression.

CHAPTER 4: VARIABLES ASSOCIATED WITH SMOKING BEHAVIOR IN THE GENERAL POPULATION

Introduction

There is a large amount of research correlating smoking cigarettes with head and neck cancer and with COPD, as well as research which attempts to predict smoking cessation. A majority of the smoking cessation literature has focused on demographic, smoking history, disease severity and disease treatment variables as predictors of smoking behavior. Although demographic, smoking history variables, and disease-related variables have been found to predict smoking cessation, these variables are static and cannot be modified. For instance, we cannot change individuals' smoking histories. However, psychosocial variables such as depression, anger and problem-solving skills, if found to be predictors of smoking behavior, are variables in which health care professionals may intervene and increase the success rate of smoking cessation within these two populations. As highlighted earlier, decreasing the prevalence of smoking in the general amd medical populations is particularly important because continued tobacco use has been shown to not only increase the risk of developing an illness, but also to increase number and severity of symptoms of disease, to reduce treatment success, and to reduce prognostic outcome.

Demographic Variables

Demographics related to smoking behavior and smoking cessation are inconclusive. The demographic variables that have been primarily studied in the smoking research include age, gender, education, and income.

Age.

Several large-scale studies have found age-related differences (Kviz, Clark, Crittenden, Freels, & Warnecke, 1994; Green, 1995b; Salive et al., 1992) in smoking behavior, whereas others have found no age-related differences (Duncan, Cummings, Hudes, Xahnd, & Coates, 1992; Matheny & Weatherman, 1998; Spitz, Fueger, Erikson, & Newell, 1990). In a cross-sectional study of 2,353 smokers, Kviz (1994) and colleagues investigated intentions to quit smoking within the next three months. Overall, less than one quarter of the sample planned to quit within the next three months, with younger smokers more likely to be "ready to quit," more concerned about the negative health effects of smoking, and more confident about their ability to quit. However, these variables discriminated between *plans* to quit rather than *actual* quitting behavior. In a community-based sample of over 10,000 adults, Salive (1992) and colleagues found that the "oldest were more likely to quit smoking and less likely to relapse" (Salive, 1992, p. 1269). Likewise, Green (1995b) found age to predict smoking cessation, with the greatest actual

decrease in smoking among individuals over 44 years of age, and the smallest decrease in cessation among individuals between 18-24 years of age. In the Framingham Heart Study, which investigated smoking behavior of adults over four decades beginning in the 1950s, older subjects, both male and female, were more likely to quit than younger subjects (Freund, D'Agostino, Belanger, Kannel, & Stokes, 1992).

Alternatively, Matheny and Weatherman (1998) conducted a clinical trial smoking cessation program with 263 individuals, and found that after completing a smoking cessation program, age did not discriminate between current smokers and former smokers. Likewise, in a large-scale study of over 5,000 respondents, there was "no clear-cut pattern between age and the prevalence of current smoking" (Spitz et al., 1990, p. 75).

Gender.

Studies on the relationship between gender and smoking behavior are inconclusive and yield mixed results. One review of the literature reported that while not all studies show gender differences, in those that do, men have been more successful at quitting (Ward, Klesges, Zbikowski, Blis, & Garbey, 1997). Garfinkel (1997) reported that since 1965, the prevalence of smoking has decreased among both men and women, with a more rapid decrease among men. Although there are several studies investigating gender differences in readiness to change (Tessaro et al., 1997), studies investigating actual smoking behavior have found no gender differences in relapse rates (Brown, 1995; Matheny & Weatherman,

1998; O'Connor, Carboni, & DiClemente, 1996). In the Framingham Heart Study, women and men were equally likely to quit smoking, with 42% of women smokers and 41% of male smokers having quit after 32 years of follow-up (Freund et al., 1992). Furthermore, amount of cigarettes smoked predicted cessation in women, but not in men, such that for every half pack fewer cigarettes smoked, women were 2.1 times more likely to quit. Finally, heavy female smokers (greater than 2 packs per day) were found to be less likely to quit than male smokers (Freund et al., 1992). In a sample of 135 smokers, gender predicted long-term (e.g., through 12 months) relapse but not short-term (15 days post cessation) such that women were more likely to relapse after controlling for other demographic and smoking history variables (Ward et al., 1997).

Alternatively, in a sample of 19,960 adults, men were more likely to quit smoking than women, and this gender difference became more evident after controlling for age, education and amount smoked (Osler, Prescott, Gidtfredsen, Hein, & Schnohr, 1999). The higher quit rate among men has been found in several other studies (Lichtenstein, Lando, & Nothwehr, 1994; McWhorter, Boyd, & Mattson, 1990; Pirie, Murray, & Luepker, 1991; Royce, Corbett, Sorensen, & Ockene, 1997; Ward et al., 1997).

Several studies have found no gender differences in either direction. Freund et al. (1992) found no difference in quit rates among men and women, except among heavy smokers (as highlighted above). Finally, gender did not predict smoking cessation at one-year follow-up in a group of medical patients (Duncan et al., 1992).

Education.

The correlation between education and smoking has yielded diverse results. In a sample of medical patients, Duncan et al. (1992) found that those with a higher education (e.g. with some college education) were more likely to quit smoking that those without a college education. Wise et al. (1997), in their workshop discussion of smoking in the United States, characterized smokers as having lower socioeconomic status and a lower educational status. In a sample of 2,462 adults, the risk of being a current smoker, as opposed to a former smoker or never smoker, was higher among lower educational groups (Stronks, Van de Mheen, Looman, & Mackenback, 1997). Garfinkel (1997) found a greater smoking prevalence among those with a less formal education. Finally, "current smoking prevalence was highest among persons with 9 to 11 years of education … and lowest among persons $\geq$ 16 years of education" (CDC, 1997, p. 1217).

Alternatively, some studies demonstrate that education does not discriminate between smokers and former smokers (Matheny & Weatherman, 1998) and does not predict smoking cessation (Freund et al., 1992). Furthermore, Green (1995a) found education to impact smoking behavior in the opposite direction such that individuals who were less-educated were "more responsive to the intervention than college-educated smokers" (p.189).

Income.

There is very little research investigating the relationship between income and smoking behavior. Spitz (1990) and colleagues found that "high-income (>$43,000) respondents were significantly more likely to be heavy smokers than respondents with the lowest incomes (<$23,000 annually)" (p.77).

Knowledge and Risk-Perception

Research results on the impact of knowledge on smoking behavior are mixed. Some studies have found that smoking status is a function of awareness of the risks from smoking. In other words, knowledge of the negative health effects of smoking has been found to be lower among smokers as compared to nonsmokers (Brownson et al., 1992; Lee, 1989). For instance, Brownson and colleagues surveyed over 2,000 adults about their beliefs about smoking. Eighty-eight percent believed smoking to be harmful to their health; with 74% reporting the knowledge that smoking causes emphysema. However, "current smokers were significantly less likely than never smokers to acknowledge the health effects of smoking" (Brownson et al., 1992, p. 100). In addition, 83% reported to believe quitting smoking has health benefits. Overall, knowledge was lower for those who were older, female, less educated or current smokers. Other studies have found that smokers and non-smokers are similarly and well aware of the health risks of smoking, yet smokers tend to underestimate

their own personal health risk when they compare themselves to other smokers (McCoy et al., 1992; Strecher, Kreuter, & Kobrin, 1995). A more recent study found that knowledge about the causes of oral cancer among current smokers, quitters and never smokers was not significantly different (Fabian, Irish, Brown, Liu, & Gullane, 1996). The researchers compared 22 patients diagnosed with oral cancer to 112 patients without head and neck cancer. Only 20 out of the 124 participants (14.9% of the total group) were aware of the causes of oral cancer (3 of the 22 cancer patients and 17 of the 112 controls) (Fabian et al., 1996). There was no statistical difference in knowledge between the groups. Furthermore, knowledge about the causes of mouth cancer between smokers, those who had quit smoking, and non-smokers was not significant" (Fabian et al., 1996, p. 90). However, approximately 86% were aware of the association between smoking and the development of lung cancer. Results did not indicate whether there was a difference in this knowledge among smokers, former smokers and non-smokers.

Tessaro (1997) and colleagues found that smokers who perceived that lung cancer would result from smoking were more likely to be thinking about quitting smoking. However, actual smoking quits was not investigated in this study.

Smoking History Variables

Overall, smoking variables have been found to predict smoking cessation and relapse. Several of the more popular ways of measuring smoking variables have included nicotine dependence, pack years, number of cigarettes per day, number of years smoked, and finally, number and/or duration of prior quit attempts.

Shiffman, Hickcox, Paty, Gnys, Kassel, & Richards (1996) found that, in a sample of 133 ex-smokers, more nicotine-dependent (defined by self-reported baseline smoking rate and a modified version of the Fagerstrom Tolerance Questionnaire) participants progressed more rapidly toward relapse. Likewise, Duncan et al. (1992) found that in a sample of medical patients, those who were nicotine-dependent (e.g. those who smoked within 15 minutes of awakening) were less likely to have quit at one year follow-up. Another study found that "heavy smoking was significantly more prevalent among current smokers than former smokers" (Spitz, Fueger, Erikson, et al., 1990, p.77). Alternatively, in a sample of 215 smokers, self-reported nicotine dependence did not predict urges to smoke (Doherty, Kinnunen, Militello, & Garvey, 1995). Relapse was greatest immediately after quitting, and relapse was highly correlated with dysphoric emotions.

A majority of studies suggest that the number of cigarettes smoked per day is a good predictor of smoking cessation success. In a summary of 10 longitudinal smoking cessation studies, light smokers (defined as smoking less than 21 cigarettes) were 2.2 times more likely to quit for 12 continuous months than heavy smokers (defined as smoking at least 21

cigarettes per day) (Cohen, Lichtenstein, Prochaska, Rossi, Gritz, et al., 1989). In a sample of 10,300 participants over 65 years of age, Salive (1992) and colleagues found that individuals who smoked fewer cigarettes or who had smoked for fewer years were significantly more likely to quit smoking at the end of three years, and moreover, a heavier smoking history was predictive of relapse within the three year period. In a treatment outcome study, Green (1995a) also found a significant decrease in prevalence of smokers in the intervention group among light- and moderate-smoker cohorts as compared to the heavy smokers. Heavy smoking also was found to predict smoking cessation in a large population sample (Osler et al., 1999).

Alternatively, Matheny and Weatherman (1998) found smoking variables, specifically tar content and number of cigarettes per day, to be predictive of maintenance of abstinence, but in the opposite direction. Surprisingly, those who had smoked cigarettes with a higher tar content, and those who had smoked a greater number of cigarettes per day, were more likely to be abstainers. Number of cigarettes per day correctly classified 85% of relapsers and 42% of abstainers. This finding that greater number of cigarettes smoker per day predicted abstention goes against previous research findings reporting that the greater number of cigarettes smoked per day predicts unsuccessful cessation outcomes (Gunn, 1983; Mothersill, McDowell, & Rosser, 1988; Ockene, Benfari, Nuttall, Hurwitz, & Ockene, 1982).

Another smoking variable found to predict future smoking cessation is number and duration of previous quit attempts. Mothersill (1988) and colleagues found that among 333

adult smokers, longer duration of previous quit attempts predicted continued abstinence. Alternatively, Cohen et al. (1989) found that the number of previous quit attempts was unrelated to successful smoking cessation.

Social Support

Several studies have investigated the role of social support in the efficacy of smoking cessation programs. Such studies have primarily focused on the effect of living with other smokers. Results of these studies concur that the existence of other smokers in the household (Matheny & Weatherman, 1986; McIntyre-Kingsolver, Lichtenstein, & Mermelstein, 1986) and exposure to other smokers (Horowitz, Hindi-Alexander, & Wagner, 1985; Wewers, 1988) affects continued abstinence. For instance, McIntyre-Kingsolver and colleagues (1986) compared smoking intervention with smokers and an intervention with smokers and their spouses. They found that the smoking status of the two groups did not differ significantly at 12 month follow-up. However, the spouses' smoking status was significantly related to the subject's smoking status such that those with nonsmoking spouses were more likely to maintain abstinence at 6-month follow-up, regardless of whether the intervention included spouses. Similarly, Matheny and Weatherman (1998) found that relapsers had a significantly greater number of other smokers in the household than did continued abstainers.

Depression

Depression has been associated with smoking in adolescent (Anda et al., 1990; Coogan et al., 1998; Wang, Fitzhugh, Westerfield, & Eddy, 1994; Wang, Fitzhugh, Eddy, & Westerfield, 1998), adult general, and adult medical populations (see Glassman et al., 1993 for review; Kick and Cooley, 1997; Parchman, 1991). Smoking has been found to be correlated with a lifetime history of Major Depression (Glassman et al., 1990) and a history of major depressive disorder among smokers has been found to be as high as 44% (Ginseberg, Hall, Reus, & Munoz, 1995), higher than the prevalence rate of MDD in the general population. Wang (1994) and colleagues found that in a sample of 6,900 adolescents (885 regular smokers and 6,015 never smokers), smokers were more likely to report symptoms of depression (through a telephone interview) such as feeling unhappy and feeling hopeless about the future. Likewise, Coogan (1998) and colleagues found that in a sample of 31, 861 adolescents in grades 4-6, 15 % (n=4,884) were smokers, with smokers more likely than nonsmokers to report stress and depression (on a survey developed by the researchers of the study). In a sample of over 1,000 young adults, Breslau, Kilby, and Andreski (1993) found that "nicotine dependence … was positively associated with … negative affect, hopelessness, and general psychological distress (p.945). Swan, Ward, and Jack (1996) found, in a sample of 64 ex-smokers, that depressed mood (as measured by the depression/dejection subscale of the Profile of Mood States) was associated with a higher rate of relapse. Likewise, Shiffman et al. (1997) found that more nicotine dependent smokers tended to lapse when experiencing negative affect (as reported by participants in an

electronic diary), and less nicotine dependent smokers tend to lapse when consuming alcohol. Other studies have also found greater negative emotion to predict stronger urges to smoke (Doherty et al., 1995; Shiffman, 1986). Overall, these studies suggest a strong association between depressive symptoms and smoking behavior.

In addition to depression being highly correlated with smoking behavior, smokers with a history of depression have been shown to be less successful at smoking cessation than nondepressed smokers (Anda et al., 1990; Ginsberg et al., 1995; Glassman, 1993; Hall, Munoz, Reuss, & Sees, 1993; Rabois & Haaga, 1997). One study found that smokers attribute their lapses to negative mood (Shiffman, Gnys, et al., 1996). Furthermore, in a sample of almost 3,000 individuals, depressed smokers (as measured by the Center for Epidemiologic Studies Depression Scale[CESD]) were 40% less likely to have quit at follow-up than nondepressed smokers (Anda et al., 1990). Research investigating adult populations have also found the prevalence of depression and depressive symptoms to be higher among smokers than nonsmokers (see Glassman, 1993 for review). In a sample of internal medicine patients, depression (measured using the Structured Clinical Interview for DSM-III-R) was highly correlated with smoking (Kick and Cooley, 1997).

Alternatively, Rabois and Haaga (1997) compared smokers and nonsmokers with and without a history of depression (according to DSM-III-R criteria) and found a main effect for depression history. In other words, participants who had a history of depression reported more negative ways of coping than did participants without a history of depression. Furthermore, there is evidence that smokers with a history of depression are less successful

at cessation than nondepressed smokers (Glassman, 1993; Hall et al., 1993). In a sample of 151 former smokers, 116 relapsed within 23 days of abstinence and 35 maintained abstinence (Shiffman, Gnys, et al., 1996). All smokers experienced temptations to quit and cravings; however, relapsers experienced greater negative affect and more cues. Likewise, in a group of 250 smokers, dysphoric emotions (as measured by the CESD) "were among the most consistent correlates of urges to smoke" (Doherty et al., 1995, p. 176). Therefore, while all faced challenges to quitting smoking, affect predicted cessation success.

Alternatively, in a sample of 613 women attending a 13-week smoking cessation program, only 18.5% met the criteria for a history of depression (Ginsberg et al., 1995) as measured by the MDD scale of the Diagnostic Interview Scale. Women who reported a history of depression were heavy smokers and had a longer smoking history. However, depression history was not associated with adherence to the smoking cessation program. Likewise, Ginsberg et al. (1997) found that "adherence to a multisession, multicomponent smoking cessation program was not found to be associated with a history of depression in women smokers who did not report any current depression (p.786).

Anger

Anger has been minimally investigated in the smoking research, and the studies which do exist demonstrate mixed findings. In some studies, anger neither classifies smokers from nonsmokers, nor distinguishes among light, moderate and heavy smokers (Muller, 1992; Witt, Kaelin, & Stoner, 1988). Muller suggests that the concept of a "smokers personality" does not exist. However, some studies show that smokers report more anger (as measured by Spielberger's State-Trait Personality Inventory and the Anger EXpression Scale) than nonsmokers (Speilberger, Foreyt, Reheiser, & Poston, 1998), and in the short term, abstinence among smokers results in increased depressive and anger symptoms (Doherty et al., 1995; Gilbert et al., 1997; Ginsberg et al., 1995; Tate, Stanton, Green, & Schmitz, 1996; Tsuda, Steptoe, West, Fieldman, & Kirschbaum, 1996). For instance, anger (as measured by the Profile of Mood States [POMS]) has been associated with a higher rate of relapse (Doherty et al., 1995). In a sample of 149 people receiving cognitive-behavioral intervention of nicotine gum, individuals with higher anger according to the Profile of Mood States "were more likely to fail to attain continuos abstinence than those with lower scores at baseline" (Hall, Munoz, & Reus, 1994, p.144). Similarly, smokers who had a history of depression reported more symptoms of anger and depression, according to the POMS, at the time of quitting than did smokers without a history of depression (Ginsberg et al., 1995). A study investigating the effects of smoking abstinence on mood in 50 male smokers, found that overall, depression and anger (according to the POMS) increased in the group of smokers who quit, and this negative affect did not return to pre-cessation levels at one month follow-up (Gilbert et al., 1997).

Coping and Problem Solving

Horowitz (1985) and colleagues found that ex-smokers "actively coped with smokers in their environment [and] avoided other smokers in public places," whereas recidivists did not engage in such coping behaviors and placed less responsibility on themselves for their health. The authors recommended coping skills training as part of smoking cessation programs. Coping style assessment revealed that smokers were less inclined to display avoidance behavior and less inclined to seek social support or to show their emotions. Wewers (1988) found that abstainers used more problem-focused coping strategies (as measured by the Ways of Coping Checklist) as compared with relapsed participants. Problem focused coping strategies included approaching a nearby smoker and asking the person to extinguish the cigarette, or leaving a stressful situation to reduce the temptation to smoke. Likewise, Matheny and Weatherman (1998) found that both stress coping resources (defined in their study as perceived confidence, physical health, physical fitness, problem solving, self-directness, and tension control and measured by the Coping Resources Inventory for Stress) and smoking history variables to be more predictive of maintenance than perceived locus of control or demographic variables.

Studies investigating relapse have suggested a multitude of background and precipitating factors such as environmental cues, biological factors such as withdrawal symptoms, personal factors such as coping ability, mood, and health status, and smoking

characteristics (see Shiffman et al., 1986 for review). Many of the studies investigating relapse rates in smoking have been guided by either a biobehavioral model, focusing on the combined psycho-biological influences on relapse, or stress-coping models, explaining smoking as a means of coping with stress. In the latter model, "high levels of stress are thought to predispose ex-smokers to relapse, but ex-smokers can be buffered from this effect if they have adequate skills other than smoking … for coping with stress" (Shiffman, Shumaker, Abrams, Cohen, Garvey, Grunberg, et al., 1986, p.21), such that coping resources may help an individual to overcome temptations to smoke during situations of high stress.

Self-Efficacy

Matheny and Weatherman (1998) found that high confidence (e.g., belief in one's coping abilities and expectations to succeed), and belief in one's self as capable and competent, predicted abstinence. In a sample of 42 smokers, followed over a one year period, Colletti and Kopel (1979) found that self-attribution (e.g. those who believed that the results of the program were due to changes in their own attitudes) was correlated with maintenance of smoking cessation. The authors suggested that this study supported the "role of cognition as predictors of maintenance of behavior change" (Colletti & Kopel, 1979, p.167). In a later study of 29 people participating in a smoking reduction clinic, Colletti, Supnick, and Payne (1985) found that "individuals who relapsed before 1-month follow-up

had lower [self-efficacy] scores that those who relapsed after 3-month follow-up" (p. 255). In a 6-month prospective study of 146 adults who attended a smoking cessation program, Baer, Holt and Lichtenstein (1986) found that self-efficacy predicted smoking rates but not smoking status. In other words, self-efficacy was not correlated with abstinence, but was related to reductions in smoking rates. Furthermore, they found self-efficacy to be a good predictor of relapse. The role of self-efficacy on improving smoking cessation success has also been found in other studies (DiClemente, Prochaska, & Gilbertini, 1985; Grove, 1993; Kowalski, 1997; Lawrence & Rubinson, 1986; Mothersill et al., 1988; Strecher et al., 1995).

Locus of Control

Bennett, Norman, Moore, and Murphy (1997) found smokers to hold stronger internal, chance and powerful others beliefs than never smokers. They also found that the interaction between health locus of control (LOC) and value for health is a significant predictor of smoking status. In other words, one's value for health moderates the relationship between health LOC and health-related behaviors (e.g., smoking tobacco). Another study found that former smokers (i.e., quitters) were significantly more internal than either current or never smokers (Molloy et al., 1997). Locus of control has been found to account for 30% of the socioeconomic gradient (Stronks et al., 1997). In other words, although there was a significant difference in smoking behavior as a function of educational level, this risk

was associated with the fact that individuals in lower SES more frequently had an external locus of control. Bunch and Schneider (1991) found that "addicted smokers scored more externally [on a measure of LOC] than nonaddicted smokers" in a group of 70 smokers. Matheny and Weatherman (1998) found that current smokers more frequently had an external locus of control as compared to never and former smokers (though the difference was not statistically significant). In a population survey, Owen and Brown (1991) found that those individuals who had made more cessation attempts in the past or who had abstained for less than a week were more likely to attribute their current smoking status to uncontrollable factors such as addiction.

Summary of Variables Related to Smoking Behavior in the General Population

Research investigating predictors of smoking behavior in the general population indicate the following: demographic variables predict smoking behavior in some, but not all, studies; knowledge and risk-perception predict smoking behavior in some, but not all, studies; social support, and in particular, exposure to other smokers, predicts smoking behavior in some, but not all, studies; smoking history variables predict smoking behavior, and depression and coping skills predict smoking behavior. There is also some evidence that anger predicts smoking behavior, but research in this area is limited.

CHAPTER 5: VARIABLES ASSOCIATED WITH SMOKING BEHAVIOR IN THE CANCER POPULATION

Introduction

This section will review the current research related to smoking in the cancer population. When possible, attention will be given to studies investigating head and neck cancer.

Demographic Variables

Age.

Studies investigating age-related smoking differences have yielded varied results. For instance, in a group of 65 women diagnosed with lung cancer, Sarna (1995) found that current smokers are more likely to be younger. Alternatively, Gritz et al. (1993) found that younger smokers quit at higher rates. Several studies have found no age-related differences. Moore (1971) found no age-related differences in smoking cessation in a sample of 203 patients with cancer of the mouth, pharynx, and larynx, or in smoking cessation. Likewise, several other studies found no age-related differences in a group of patients with head and

neck cancer (Ostroff et al., 1995; Schnoll et al., 2002; Spitz, 1995; Vander Ark, DiNardo, & Oliver, 1997).

Gender.

Studies investigating gender-related differences in smoking in the cancer population reveal mixed results. Moore (1971) found no gender-related differences in smoking cessation. In a sample of 160 patients with head and neck carcinoma, "more than half the patients continued to smoke, with the women patients being slightly more reluctant to give up their habit" (Silverman et al., 1983, p.34) at one-year follow-up. A recent study of 75 patients with head and neck cancer and lung cancer also found no age-related differences (Schnoll et al., 2002). Alternatively, Vander Ark (1997) and colleagues found that men were less likely to quit (61%) than women (82%), although the authors of this study did not report whether this difference was statistically significant given the variability of the distribution (70 males and 17 females).

Education.

Ostroff (1995) and colleagues found that those who were less educated were more likely to abstain as compared to individuals with higher education, who were more likely to relapse. Alternatively, Vander Ark (1997) and colleagues identified a trend such that "those with a high school education or greater were more likely to achieve smoking cessation" (p. 890). Schnoll et al. (2002) found no education-related differences.

Income.

Vander Ark (1997) and colleagues found that those who earned less than $10,000 per year were more likely to quit than those who earned $10,000 or more. However, the author did not report if the difference between these two groups was significant. Schnoll (2002) and colleagues found no differences between smokers and abstainers on income.

Ethnicity.

One study found that minority smokers quit at higher rates than nonminority smokers (Gritz, Carr, et al., 1993). Other studies did not report on ethnicity differences (Ostroff et al., 1995; Vander Ark et al., 1997).

Disease Severity and Treatment Variables

Overall, disease severity and treatment severity have been associated with cessation success.

Gritz, Carr, et al. (1992) conducted a smoking cessation intervention study with 186 current and recent smokers with first primary squamous cell carcinoma of the upper aerodigestive tract. They found that 88 percent of the patients were current smokers and at 12 month follow-up of a smoking cessation program, 70.2% of the participants were

complete abstainers (Gritz, Carr, et al., 1993). One month follow up revealed a majority of the participants quit smoking prior to or at initial advice. One of the predictors of continuous abstinence was medical treatment. More specifically, patients receiving primary radiation therapy achieved a low 36% abstinence as compared to patients who had undergone a total laryngectomy, who achieved an 87% abstinence rate.

Ostroff (1995) and colleagues investigated prevalence and predictors of continued tobacco use after treatment of patients with head and neck cancer. Specifically, these authors were interested in the role of disease and treatment characteristics in continued tobacco use. One hundred forty-four patients who had undergone surgical treatment for newly diagnosed head and neck cancers at Memorial Sloane-Kettering Cancer Center between January of 1991 and June of 1992 completed a tobacco-use survey. Smoking status was based on even a single puff during the time period specified. Thirty-five percent (26 out of 74) of those who had smoked the year before diagnosis (or 18% of the total sample) reported continued tobacco use after surgery. Of these 26 patients, 88% believed quitting smoking would be beneficial, 92% had thought about quitting, and 84% had made at least one attempt to quit. Those more likely to abstain from smoking had undergone postoperative radiotherapy, had cancers of the larynx or pharynx as opposed to cancer of the oral cavity, and had more severe disease. For instance 83% of patients who underwent postoperative radiation treatment remained abstinent versus 53% of the patients who did not receive post-operative radiation treatment. Also, “72% of patients with Stage II-IV disease were likely to have remained abstinent, compared to 24% of patients with in situ or Stage I

disease" (p.573). Moreover, in this study, of those who relapsed, resumption typically occurred one month after surgery and of those who returned, 58% were daily users. Continued use of tobacco was associated with less severe disease and less extensive treatment. Furthermore, although state of disease and post-operative radiotherapy were the best predictors of continued smoking, these disease variables did not account for a significant additional variability in smoking status after disease site.

Likewise, Vander Ark (1997) and colleagues found disease and treatment severity to be predictive of smoking cessation in a group of 87 patients with squamous cell carcinoma of the head and neck. Overall, 69% of the participants quit smoking. Seventy-five percent of those who did quit did so because of a diagnosis of cancer. Forty percent quit at the time of diagnosis, 19% at time of treatment, and 16% after therapy. Combined therapy (e.g., surgery and post-operative radiation) was a positive predictor of smoking cessation as was a total laryngectomy. For instance, 95% of patients who underwent a total laryngectomy and post-operative radiation therapy quit smoking as compared to 47% of the patients who received radiation therapy alone or 63% of the patients who had undergone the surgery without postoperative radiation therapy. Ostroff et al. (1995) reported similar findings such that abstainers were those who had more extensive disease. More specifically, "72% of patient with stage II-IV disease were likely to have remained abstinent, compared with 24% of patients with in situ or Stage I disease" (p.573). Furthermore, "patients with less extensive disease were two times more likely than patients with more extensive disease to resume tobacco" (p. 574). In a recent study of 75 patients with either head and neck cancer

(n = 23) or lung cancer (n = 51), when time since diagnosis was partialled out, "patients who were not undergoing treatment were more likely to be smokers (Schnoll et al., 2002, p. 140).

Smoking and Alcohol Variables

Smoking behaviors and risk factors were examined in a sample of 5,998 cancer patients between February 1986 and February 1988 (Spitz, Fueger, Eriksen, et al., 1990). Smoking history was defined as "smoking cigarettes on a daily basis for at least 6 months" (p. 74). Approximately 30% of the participants were current smokers. There was no clear-cut pattern between age and the prevalence of current smoking. However, heavy smoking was significantly more prevalent among current smokers of both genders than among former smokers, suggesting that heavy smokers may have more difficulty quitting.

Vander Ark (1997) and colleagues found prior alcohol use to be a predictor of smoking cessation, as did Duffy et al. (2002) in a sample of 81 head and neck cancer patients. It has also been found that heavy smokers have a more difficult time quitting than light or moderate smokers (Gritz, Carr, et al., 1993). This study also found that nicotine-dependent smokers, defined as "time to first cigarette" (p. 268), have more difficulty with cessation than non-nicotine dependent smokers. Recent studies have similarly found that smokers tend to be more dependent on nicotine than abstainers (Duffy et al., 2002; Schnoll et al., 2002).

The role of tobacco, in combination with alcohol use, has also been investigated. Ostroff (1995) and colleagues found that the number of years a person smoked and daily consumption of alcohol was not associated with continued tobacco use. A study of 545 cases and 641 controls (referents) investigating the use of tobacco, oral snuff and alcohol in the etiology of squamous cell carcinoma of the head and neck found that there was a fourfold increased risk for cancer for ever-users (Lewin et al., 1998). This risk increased with duration of smoking, not intensity. Of the cancer cases, 44 (8%) had never smoked and 501 (91.9%) had a smoking history, 385 (76.8% of ever smokers) were currently smoking, and 116 (23.1%) were ex-smokers. They found that alcohol had increased the relative risk of head and neck cancer for smokers but not for nonsmokers. In fact, the combination of alcohol and tobacco produced a multiplicative effect on the relative risk, which has also been found in other studies (Bundgaard, et al., 1995).

Several studies have shown that alcohol interacts with smoking, contributing significantly to the development of oral cancer (Blot et al., 1998; Bundgaard et al., 1995), pharyngeal cancer (Blot et al., 1998), an increased risk of second cancers (Day et al., 1994), and an increased risk of death (Deleyiannis, Thomas, Vaughan, & Davis, 1996). Deleyiannis et al. (1996) investigated smoking behavior in 649 patients diagnosed with cancer of the oral cavity, oropharynx, hypopharynx, and larynx, and found that alcoholism "was associated with increased risk of death …. independent of age, site of cancer, anatomical stage, histopathology, grade, smoking and type of antineoplastic treatment" (p.542). For instance, after adjusting for smoking variables, the 5-year survival estimate for

abstinent alcoholics and for alcoholics currently drinking was 57.1% and 40.9%, respectively (Deleyiannis et al., 1996), and the authors suggested that quitting alcohol use may prolong life. Furthermore, there was an association between decreased alcohol use and decreased smoking such that 55.3% of abstinent alcoholics versus 73.7% of nonabstinent alcoholics were currently smoking 20 or more cigarettes per day (Deleyiannis et al., 1996).

Likewise, prior heavy alcohol use was found to be a negative predictor of smoking cessation (Vander Ark et al., 1997). "The risk of esophageal cancer decreased rapidly, strongly, and significantly with longer periods of abstention" of both tobacco and alcohol use (Castellsague et al., 2000, p. 814). The authors reported that that decreased use of both tobacco and alcohol decreased the risk of developing esophageal cancer, "regardless of amount, duration, or type of tobacco and alcoholic drink consumed" (Castellsague et al., 2000, p.817).

Psychosocial Variables

Many studies have indicated that depression is prevalent among individuals with cancer (Derogatis et al., 1983; see Massie and Popkin, 1998 for review). Research suggesting a high correlation between depression and smoking behavior in the general population was highlighted earlier. In a sample of over 1,100 patients with cancer, approximately 35% reported mild to severe depression according to the Zung Self-Rating Depression Scale (Passik et al., 1998). According to Massie and Popkin's (1998) review,

depression among cancer patients ranges from 1% - 53%, depending on the study. Furthermore, among individuals with cancer, advanced stages of disease correlated with a higher prevalence of depression. These high prevalence rates are important to consider, since in the general population, smokers with depression are less successful at cessation. Given the data in the general population, depressive symptoms may predict smoking behavior in the cancer population as well. To date, however, depression in the context of smoking cessation among cancer patients has bee examined in only one study (Schnoll et al., 2002), in which smokers displayed higher levels of emotional distress as measured by a revised impact of events scale ([RIES]).

CHAPTER 6: VARIABLES ASSOCIATED WITH SMOKING BEHAVIOR IN THE COPD POPULATION

Introduction

This section will review the current research related to smoking in the COPD population.

Demographic Variables

Age.

Overall, studies conducted by Pederson and colleagues have found that successful quitters tended to be older than those who continued to smoke, although this difference was not significant (Pederson, Wanklin & Lefcoe, 1991). Another study also found that older patients were more likely to quit than younger patients (Bjornson et al., 1995). Wise (1997) found that "there have been substantial reductions in smoking behavior in young adults" (p. 420), whereas "older groups have shown little or no reduction in smoking prevalence" (Wise, 1997).

Gender.

The predictive role of gender and smoking behavior in the COPD population has yielded mixed results. Some studies have demonstrated that gender is associated with smoking cessation (Pederson, Baskerville, & Wanklin, 1982; Pederson, Williams, & Lefcoe, 1980) whereas a more recent study did not demonstrate a relationship (Pederson et al., 1991).

In the Lung Health Study, 27% (n=1069) of all participants in the Special Intervention Group were sustained nonsmokers at 12 months (Bjornson et al., 1995). Results showed that men had a higher sustained quit rate at 12 and 36 month follow-ups. Variables that were related to men's longer abstention rate were education, length of longest quit attempt, having another smoker in the household, consuming more than 7 drinks per week, smoking more than 30 cigarettes per day at baseline and finding the first cigarette for the day the hardest to give up. Overall, women had greater difficulty quitting smoking than men. However, gender alone explained only some of the difference in smoking cessation rates. Within each gender group, those who were "better educated, married, older, had made longer quit attempts in the past, had not used Nicorette before, … and had a lower cotinine level were more likely to be sustained nonsmokers" (Bjornson et al., 1995, p.228).

Education.

Some studies have found a relationship between educational level and smoking behavior (Bjornson et al., 1995; Pederson et al., 1982; Pederson et al., 1980), while others have not found an association (Pederson et al., 1991). For instance, in the Lung Health Study, those who were more educated were more likely to be sustained nonsmokers (Bjornson et al., 1995). Pederson et al. (1980) found that the older and younger COPD patients were more likely to quit smoking than the middle-aged patients.

Income.

Socio-economic status has been associated with smoking cessation in some studies (Pederson et al., 1982; Pederson, Wanklin, & Baskerville, 1984; Pederson et al., 1980; Pederson et al., 1988) but not in others (Pederson et al., 1991). For instance, Pederson et al. (1980) found that individuals in the upper and middle social status were more likely to quit smoking as compared to individuals in the lower social status. Alternatively, future smoking status was not related to socioeconomic status (Pederson et al., 1991).

Ethnicity.

Although there has been a "greater proportional decline in smoking prevalence among African-Americans than among whites," there is still a greater prevalence of smoking among African-American men" (Wise, 1997, p. 420).

Disease Severity Variables

In a sample of 45 patients with COPD, severity of disease (defined by FEV1) was not related to initial or follow-up smoking status (Devins & Edwards, 1988). Other studies have found a higher smoking cessation rate with increased severity (Pederson et al., 1980). However, given the chronicity of the COPD, many patients may have tried repeatedly to quit smoking over the course of their disease (Orleans et al., 1993).

Smoking and Alcohol Variables

Murray, Istvan, Voelker, Rigdon, and Wallace (1995) reported on the association between alcohol consumption and smoking cessation as part of a large scale study, the Lung Health Study. The Lung Health Study was a randomized multi-center clinical trial sponsored by the Division of Lung Disease of the National Heart, Lung, and Blood Institute. The goal of this study was to determine whether an intensive smoking cessation program in combination

with an inhaled bronchodilator could reduce the rate of decline of pulmonary function and thereby reduce respiratory morbidity in middle-aged smokers with mild airflow obstruction over a 5-year period. Participants included smokers ages 35 to 60 who were at the early stages of COPD. Murray (1995) and colleagues found that, in a sample of 3,997 men and women in the Lung Health Study, nearly half of all participants quit smoking. Only baseline binge drinking predicted smoking behavior at one year follow-up. Neither amount of drinking nor drinking status were predictive of smoking status after one year for the intervention group. However, in the control group, baseline nondrinkers smoked more heavily than drinkers after one year. Measures of smoking status were assessed via self-report, carbon monoxide in expired air and salivary cotinine. In the intervention group, those who had quit smoking were heavier drinkers than those who did not quit smoking. According to these authors, this finding suggested that individuals with two habits will quit drinking alcohol before quitting smoking cigarettes. The authors did not find consistent results regarding the relationship between alcohol and relapse.

Crowley, MacDonald, and Waller (1995) implemented a smoking cessation program with 49 smoking COPD patients. Individuals in the experimental group were reinforced for smoking cessation with lottery tickets whereas individuals in the control group received non-contingent lottery tickets (i.e., received lottery tickets that were not contingent upon co-validated smoking status). At 6 month follow up, 31/36 (90%) patients were still smoking and 3 individuals (10%) were no longer smoking. Overall, few patients quit smoking, and quit rates between the experimental and control groups did not differ

significantly. Advice and reinforcement, including money, were not enough of a reinforcement to quit smoking. Patients pretreatment reports of desire to quit did not predict outcome, nor did scores on "dependence."

Depression

Several studies have demonstrated a correlation between depression and COPD (Agle, Baum, Chester, & Wendt, 1973, Light, Merrill, Despars, Gordon, & Mutalipassi, 1985; Weaver & Narsavage, 1992). In a sample of patients with moderate to severe COPD, 42% had significant depression (Light et al., 1985). Weaver and Narsavage (1992) found that approximately half of their participants attending an outpatient clinic for COPD had a mean depressive mood score (as measured by the Multiple Affect Adjective Check List Revised [MAACL-R) greater than norm, and this was significantly correlated with functional status. Unfortunately, no studies were found which specifically examined the relationship between depression and smoking in the COPD population.

Self-Efficacy

Research on smoking cessation within the COPD population shows that overall, COPD patients do not quit smoking. An early study investigating the role of self-efficacy in smoking cessation among 45 COPD patients found only 11.1% quit at 3 month follow-up

(Devins & Edward, 1988). However, 68 % significantly reduced their number of cigarettes per day. Perceived self-efficacy was a significant predictor of smoking behavior change.

CHAPTER 7: MEASUREMENT OF SMOKING VARIABLES

Velicer, Prochaska, Rossi, and Snow (1992) reviewed various self-report and biochemical measures of smoking status, as well as issues to consider in choosing a smoking measure for research, including type of study, type of population, and demand characteristics. Three ways to measure self-reported smoking status include point-prevalence, continuous abstinence and prolonged abstinence. Overall, all of these measures are "ad hoc measures" (i.e., "do not reflect the complex variety of changes that are involved in smoking cessation" (Velicer et al., 1992, p.35).

Point prevalence, which will be utilized in this study, is defined as the "percentage of smokers who are not smoking at the particular point in time" (Velicer et al., 1992, p. 23). Point prevalence self reports have the advantage of including individuals who were "delayed quitters." Point prevalence rates "also allow lapses or relapses to occur following treatment without making it necessary to categorize the smoker as a permanent failure" (Velicer et al., 1992, p.26). It thereby recognizes the dynamic nature of the smoking/not smoking process. However, such self reports may overestimate cessation by including short-term quitters who have not yet "had time" to relapse. Furthermore, because of high relapse rates within the first three months following quitting … many individuals who are counted as former smokers at one point in time will be current smokers at the next point in time" and "abstinent rates are not stable" (Velicer et al., 1992).

Three ways to obtain smoking status are through self-report, report by significant others, or use of biochemical markers, such as measures of carbon monoxide, thiocyanate, and cotinine. The general advantage to self reports is the greater ease and lower cost in collecting data. However, biochemical verification reduces misreporting (Velicer et al., 1992) among populations which may feel high demand characteristics to underreport. Several studies have shown a higher rate of false negatives for medical populations (Sillett, Wilson, Malcolm, & Bal, 1978; see Velicer et al., 1992, for review; Vogt, Selvin, Widdowson, & Hulley, 1977) than in other populations. The use of biochemical markers, however, is not a cure-all to the underreporting problem. The half lives of CO, thiocyanate, and cotinine are 5 hours, 10-14 days and 15-40 hours, respectively. Therefore, biochemical markers can only assess for recent smoking, going as far back as up to one month prior to the assessment. Furthermore, a disagreement between a self-report and a biochemical marker may not only reflect underreporting, but may also reflect environmental conditions (e.g., individuals who work with certain chemicals). In addition to disadvantages of measurement, the use of biochemical markers may increase refusal rate of participation in the studies.

In addition to biochemical validation, another method sometimes used to decrease underreporting or false negative rates is to introduce the "bogus pipeline" (Evans, Hansen, & Mittelmakr, 1977; Jones & Sigall, 1971). As part of this procedure, subjects are told that biochemical assessments will be used to assess smoking status, when they will actually be collected, but not evaluated (Velicer et al., 1992, p.32). Murray, O'Connell, Schmid, and Perry (1987) provided an extensive review of this procedure and found that half of the

studies investigating the bogus pipeline effect found no difference in self-reported smoking rates between those assessed under the pipeline versus no pipeline procedures, while half of the studies did find significant differences in reporting. Most of these studies were conducted with adolescent students, which represent a very different population from adult medical populations. In addition, several of these studies showed that the students, unlike medical patients, did not experience pressure to underreport. Finally, the studies did not test whether the participants actually believed in the bogus pipeline. Murray and Perry (1987) found that among a group of 770 adolescents, simply ensuring anonymity reduces underreporting and therefore reduces the need for the bogus pipeline procedure. Among a group of 149 volunteers in a smoking cessation study, Baer et al. (1986) found that CO measures contradicted self-reported smoking status in only 3 instances.

There is some controversy over the measurement of smoking variables. Some research shows that self-reports tend to underestimate smoking habits (see Velicer et al., 1992, for review) and that people often underreport the amount they currently smoke (Murray & Perry, 1987; Stookey, Katz, Olson, Drook, & Cohen, 1987; see Velicer et al., 1992, for review), while others do not show this to be the case (Duncan et al., 1992; Glynn, Gruder, & Jegerski, 1986; Hall, Tunstall, Rugg, Jones, & Benowitz, 1985; Murray, Connett, Lauger, Voelker, 1993). For instance, in a sample of over 2,000 medical patients, 245 out of 334 (75%) self-reported quitters passed biochemical validation (Duncan et al., 1992). In a study of 127 adult smokers participating in a smoking cessation program, "[o]nly 16% of self reports of abstinence were not verified by CO measurement (Glynn et al., 1986, p.125).

Furthermore, there was no significant difference in misreporting between the groups who were educated about CO validation and informed that they would be tested, and the control group (who was not educated and informed about CO validation techniques). Thus, the authors suggest that a biochemical index "is not indicated to validate self-reports" (p. 134). In a group of 120 individuals undergoing a smoking cessation program, "biochemical measures failed to verify self report in only 3 instances (Hall et al., 1985, p. 256). Among those participating in the Lung Health Study, 1% of individuals in the usual care group reported not smoking but had positive cotinine, and 6% of those in the intervention group underreported based on a cotinine assay (Murray et al., 1993). While these differences were found to be significant, the effect size was small and the authors suggest that the size of the difference would not mislead the findings. Overall, research on false reporting of abstinence is variable (see Glynn et al., 1986 for review). In addition, several researchers have found significant inaccuracy of biochemical validation techniques (Abrams, Follick, Biener, Carye, & Hitti, 1987; Petitti, Friedman, & Kahn, 1981; see Velicer et al., 1992, for review), suggesting that the cost of such techniques does not outweigh the gains and does not provide significant additional, nor necessarily more accurate, information. For instance, saliva has a false positive rate "in periods less than seven days of abstinence" (Abrams et al., 1987).

Overall, point prevalence measures are the most popular in survey research studies investigating the relationship between smoking and disease (Velicer et al., 1992). However, research has not been consistent on how to measure point prevalence rates. Common minimum periods of abstinence are 24 hours, 7 days and 30 days. According to Velicer et

al. (1992), the National Interagency Council for Smoking and Health has recommended using a minimum of 7 days of abstinence for defining cessation, which has also been recommended by several other authors (Lichtenstein & Glasgow, 1992; Velicer et al., 1992). Several studies have defined current smokers as anyone who has smoked in the past 7 days (Duncan et al., 1992; Ostroff et al., 1995). Classification rates of smokers, quitters, and relapsers will depend on how liberal the criterion is. According to the U.S. Department of Health and Human Services, using a 1-day quit criteria, 18% will quit and 70 to 80% will relapse, whereas using a 7-day criteria, only 12% will have attempted to quit, and only a minority will relapse (Velicer et al., 1992).

In addition to biochemical validations of self-report, a less expensive validation technique to verify self reports is with an informant who lives with the participant or has regular contact with the participant (Ossip-Klein et al., 1991). Measures that have been developed to assess nicotine dependence (e.g., Fagerstrom Test for Nicotine Dependence) have yielded mixed findings with respect to it psychometric properties. However, "time to first cigarette" has been found to be a valid measure of nicotine dependence because in the morning, blood nicotine levels have decreased, and presumably, dependent smokers will experience withdrawal upon waking and smoke as soon as possible to relieve these withdrawal symptoms. Correlations between self-report measure of dependence and biochemical measures have been shown to be high (Hall et al., 1985). Overall, Velicer et al. (1992) concluded that "false reporting does not support the regular use of biochemical

markers," mostly because underreporting is relatively low, and biochemical markers are costly and difficult to obtain.

CHAPTER 8: SUMMARY OF SMOKING RESEARCH

Overall, research investigating predictors of smoking behavior has yielded disparate results. The most common variables that have been measured include demographic variables (e.g., age, gender, educational attainment, income, and ethnicity), smoking history, alcohol history, and social support. Most studies have found smoking history to be associated with smoking behavior, and most studies have found that social support is associated with smoking behavior when the measurement of social support is one which accounts for the number of other smokers in the household. Research has also examined the role of depression, anger and coping skills with respect to smoking behavior. Many of the studies have demonstrated a strong association between depression and smoking behavior (e.g., initiation of smoking cigarettes, smoking cessation, relapse) in the general population, but this relationship has been minimally investigated in the head and neck cancer and COPD populations. Approximately 70% of individuals with COPD continue to smoke as compared to approximately 30% of individuals with head and neck cancer. Continued smoking has important implications for treatment outcome, morbidity, and mortality. Much of the research in these two medical populations has focused on demographic variables, smoking history, disease severity and treatment variables, with little attention paid to psychosocial variables. In studies that do examine psychosocial variables, the variables are not examined in the context of smoking behavior, thus leaving several potential and fruitful areas for research.

CHAPTER 9: RELEVANT THEORIES

Introduction

Many theories exist which explain health behavior and which guide research attempting to understand and explain human health behavior. Several theories which have guided research on smoking behavior include the Health Belief Model (HBM), the Transtheoretical model (a.k.a. Stages of Change), and the Theory of Self-Efficacy. Although the present study will not test these theoretical constructs, the following section will describe these theories and discuss their relevance to smoking research. Finally, the Social Problem-Solving Model will be introduced, and the relevance of this model to smoking research, and this study, will be discussed.

The Health Belief Model

The Health Belief Model (HBM) is a widely used model explaining a person's health behaviors, including both behavior change and health. The HBM was initially proposed to help explain why individuals do not participate in health prevention programs (See Strecher & Rosenstock, 1997 for review). More recently, with more people living longer and in the face of chronic illness, the HBM has also been utilized to explain people's response to a

diagnosed illness (Strecher & Rosenstock, 1997). Therefore, although initially developed to help explain screening behaviors, the HBM

also explains preventive behaviors, illness behaviors and sick-role behaviors (Janz & Becker, 1984).

Simply stated, the HBM represents a value expectancy theory positing that one's behavior is not simply the result of learning, but rather, "a function of the subjective value of an outcome and of the subjective probability, or expectation, that a particular action will achieve that outcome" (Strecher & Rosenstock, 1997). Within the context of health behaviors, value can be defined as "the desire to avoid illness or to feel well" and expectancy can be defined as "the belief that a specific health action available to a person would prevent (or ameliorate) illness" (p.43).

There are four original components to the HBM: perceived susceptibility and perceived severity (which, together, comprise perceived threat); perceived benefits, and perceived barriers. Perceived susceptibility is one's perception of his or her risk of contracting an illness or, in the case of individuals already diagnosed with an illness, "acceptance of the diagnosis, personal estimates of resusceptibility, and susceptibility to illness in general" (Strecher & Rosenstock, 1997, p.44). Perceived severity measures one's perception of the seriousness of contracting an illness, or leaving it untreated, including perceptions of the medical and social consequences. Perceived benefits represents one's perceptions of the effectiveness of one's available actions to reduce susceptibility or severity. Finally, perceived barriers represent a cost-benefit analysis of the expected benefits from a health action to the expected barriers. Taken together, an individual will perform a health behavior if he or she believes the personal risk to developing an illness is high, that such an

illness will have severe consequences (e.g., pain, death, decrease in quality of life), that an available course of action will be effective in reducing susceptibility and finally, that the benefits to performing the action outweigh the barriers.

The HBM may be useful in explaining an individual's decision to continue to smoke or to quit smoking as a result of being diagnosed with cancer or COPD. This model would propose that quitters are more likely to believe that they are susceptible to worsening of symptoms, to future hospitalizations for respiratory failure or complications, to treatment failure or to recurrence. According to this model, quitters would be more likely to believe that quitting smoking will reduce their chances of disability, improve their quality of life, and reduce their chances of death. Quitters would believe that quitting smoking will, in fact, be effective in reducing their own personal susceptibility to the above negative consequences, and that the benefits of stopping smoking (e.g., reducing the risk of recurrence; reduced cost of buying cigarettes, reinforcement from family) outweighs the barriers to stopping smoking (e.g., withdrawal symptoms, weight gain, fear of increased stress). Fourth, quitters would be more likely to hold the view that the benefits to quitting outweigh the barriers. According to Strecher and Rosenstock (1997), a cost-benefit analysis of the pros and cons of smoking are "predictive of movement through stage of behavioral change readiness." Overall, the theory suggests that individuals' perceptions of personal health risk motivate health behaviors.

A summary of research on the HBM suggests some empirical support for the

model (Janz & Becker, 1984). Overall, these authors found perceived barriers to be the most powerful predictor and perceived severity to be the least powerful predictor, across various studies and behaviors (Strecher & Rosenstock, 1997). One of the main challenges to studying the application of the HBM to behaviors is that no reliable or valid measure of the HBM concepts exists (Strecher & Rosenstock, 1997).

According to Strecher and Rosenstock, the HBM is not widely used in the smoking research because "of consistent findings that the majority of cigarette smokers already perceived a general health threat from smoking" (Strecher & Rosenstock, 1997, p.50). However, although few studies in the smoking literature have tested the HBM, several studies do exist investigating the role of the HBM in explaining smoking behavior. Overall, smokers do perceive a greater threat to their health than nonsmokers (Brownson et al., 1992). In this survey of over 2,000 adult smokers, 83% of the current smokers believed smoking was harmful to their health. The fact that smokers already perceived their health as threatened makes this particular construct of the HBM irrelevant. Furthermore, this perceived threat, in essence, had no effect on their smoking behavior, suggesting that there be another underlying construct at work. It has been suggested, particularly in the smoking literature, that self-efficacy is a barrier component in the HBM (Rosenstock, Strecher, & Becker, 1988), and that this construct will be predictive of smoking behavior change among individuals with a strong perceived threat.

Some research on perceived benefits and barriers to cigarette smoking indicates that smokers and nonsmokers do differ in their beliefs that quitting smoking would result in a

health benefit (Brownson et al., 1992). In a sample of over 2,000 adult current smokers, former smokers and never smokers, they found that 8% of current smokers believed smoking was harmful to their health versus 91% of never smokers and 92% of former smokers who believed smoking to be harmful (Brownson et al., 1992). Alternatively, in a study of 308 patients recently diagnosed with pulmonary disease, Pederson et al. (1984) found that health beliefs were not associated with smoking cessation.

However, research has also shown that although smokers accurately assess the threat of smoking, they often underestimate their own personal susceptibility (Strecher et al., 1995; Weinberger, Greene, Mamlin & Jerin, 1981). Weinberger (1981) and colleagues found that both ex-smokers and current smokers viewed smoking as a threat to health, yet only ex-smokers perceived themselves to be more personally susceptible to the negative consequences of smoking than smokers. The authors concluded that in addition to believing smoking to be a serious health problem, individuals must see themselves as personally susceptible to adverse affects in order to quit (Weinberger et al., 1981)." Likewise, in a sample of 2,785 adults, smokers were more likely than nonsmokers to perceive a higher personal risk of heart attack, cancer and stroke, yet tended to underestimate their actual risk of these conditions (Strecher et al.,1995). The theory helps to explain perceived health risks in the general population.

Transtheoretical Model (Stages of Change)

The Transtheoretical Model (TM; Prochaska and DiClemente, 1983) posits that behavior change occurs through a series of linear stages. Initial studies of the TM focused on smoking behavior, and more recently, on alcohol and substance use, anxiety and panic disorder, eating disorder, HIV/AIDS prevention, mammograms, and compliance with medical regimen. The six temporal dimensions of the model include precontemplation, contemplation, preparation, action, maintenance, and termination. In the precontemplation stage, individuals are not considering taking action in the foreseeable future, typically measured as the next 6 months. The contemplation stage characterizes people who intend to change within the next six months. These individuals are often in the midst of weighing the pros and cons of behavior change. Individuals in the preparation stage intend to take immediate action, typically measured in the next month, and already have a plan of action. Finally, people in the action stage have made behavior changes in the past 6 months. In the TM, however, only total abstinence from smoking counts as a behavior change (Prochaska, Redding & Evers, 1997). The fifth component of the model is Maintenance, or the attempt to prevent relapse. The sixth and final stage is termination, characterized by individuals who have no temptation and 100% self-efficacy (Prochaska et al., 1997).

Additional constructs deemed important in the process of change are decisional balance, defined as an "individual's relative weighing of the pros and cons of changing" and self-efficacy, defined as "the situation-specific confidence people have that they can cope

with high-risk situations without relapsing to their unhealthy or high-risk habits" (Prochaska et al., 1997, p.65).

There is a tremendous amount of literature on the use of the TM with smoking and more specifically, with smoking cessation. Supporters of the TM assumes that cessation programs have not been effective because they do not take into account individual differences regarding stages of change. For instance, "people with precontemplation should not be treated as if they were ready for action interventions" (Prochaska et al., 1997). One study found that smokers in the precontemplation stage abstined the least over 18 months as compared to smokers in the preparation stage, who proressed the most (Prochaska, DiClemente, & Norcross, 1992), suggesting that knowing what stage an individual is in will allow cessation programs to be tailored more toward the individual, thereby improving success rates. Several variables, such as self-efficacy, have been shown to mediate the stage of change. For instance, self-efficacy increases as one progresses through the stages of change (Orleans et al., 1993).

A second variable important to consider is the perceived severity of the health hazard. Becker (1977) proposed that motivation to change one's health behavior depends on both the person's feelings of personal vulnerability as well as his or her beliefs about the seriousness of the health hazard. This difference in perceived hazard may explain the difference in smoking cessation rates between head and neck cancer and COPD populations. Even with the many advances in treatment for cancer, individuals with cancer may still perceive their illness to be life-threatening, whereas individuals with COPD may

experience a range of respiratory symptoms, even to the extent of a decrease in physical functioning, yet not perceive their lives to be in danger.

Although the TM may be helpful in explaining why some individuals are successful in a smoking cessation program and others are not, the TM, although important in theory, is not necessarily relevant to this study, and will not be measured specifically in this study. More specifically, a proportion of individuals participating in this study have already quit smoking. Therefore, according to the TM theory, these individuals are already in the action phase.

Theory of Self-Efficacy

Bandura (1977), in his seminal article on the impact of self-efficacy on behavior, found that behavior change is a function of one's expectation of personal efficacy. In other words, cognitive processes mediate change. Bandura's theory of self-efficacy posits that an individuals' expectations of his or her efficacy, "the conviction that one can successfully execute the behavior required to produce the [desired] outcome" (Bandura, 1977, p.193) will predict whether "coping behavior is initiated, how much effort will be expended, and how long it will be sustained in the face of obstacles and aversive experiences" (Bandura, 1977, p. 191). For instance, a person may believe that smoking is poor for his or her health. But someone who is diagnosed with a chronic condition may not believe that quitting smoking can improve an already bad situation. Another component of self-efficacy, therefore, is not only whether a particular behavior will produce the desired outcome, but

whether one can perform such behaviors (e.g., whether one can or cannot quit smoking). One might think that previous quit attempts would influence one's self efficacy to quit (e.g., "I tried to quit before and failed, so I won't be able to quit this time"). However, "expectation alone will not produce desired performance if the component capabilities are lacking" (Bandura, 1977, p.194). In other words, in addition to having a high self-efficacy, one must also have the actual coping skills to perform the desired behavior. Furthermore, one must have "incentive" to perform the behavior. Individuals diagnosed with head and neck cancer may have higher incentive because cancer is still considered a life-threatening illness whereas individuals with COPD may live many years, and experience a more gradual increase in symptoms, therefore not having incentive to quit smoking.

According to Bandura, self-efficacy is based on "four major sources of information: performance accomplishments, vicarious experience, verbal persuasion, and psychological states" (Bandura, 1977, p.195). For instance, prior success (e.g., quit attempts) increases feelings of self-efficacy whereas prior failures reduces such feelings. As another example, high emotional arousal may affect one's self-efficacy such that "diminishing emotional arousal can reduce avoidance behavior" (Bandura, 1977, p.199).

More recently , Bandura's theory of self-efficacy has been applied in research with people with cancer (see Lev, 1997, for review). More specifically, several studies have investigated the role of self-efficacy in smoking cessation. Yates and Thain (1985) found that self-efficacy successfully classified smokers into abstainers and relapsers. In a prospective study by the same authors, self-efficacy accounted for approximately 13% of the total

variance on relapse/nonrelapse (Yates & Thain, 1985). More specifically, participants' self-reported self-efficacy predicted smoking status at 8-month follow-up, suggesting that self-efficacy may serve to identify individuals at risk for relapse, and that "enhancing self-efficacy when it is low decreases the risk of relapse" (O'Leary, 1985). Finally, Utz, Shuster, Merwin, and Williams (1994) and colleagues, in a community intervention program, found that effective programs were ones which enhanced self-efficacy.

The cross-sectional design of this study does not lend itself to an examination of the influence of self-efficacy on smoking behavior. It is important to recognize that not only does an increased self-efficacy increase the likelihood of attempting to quit smoking, but a successful quit attempt may increase one's self-efficacy.

Social Problem Solving

Problem solving is "the self-directed cognitive-behavioral process by which an individual, couple, or group attempts to identify or discover effective solutions for specific problems encountered in everyday living" (D'Zurilla, Nezu, & Maydeu-Olivares, in press). It is the process by which individuals make conscious efforts to change the problematic situations themselves, their reactions to them, or both (Nezu, 1987; Nezu, Nezu, & Perri, 1989). In this model, problems are defined as "specific existing or anticipated situations that demand responses for adaptive functioning, but are not met by effective coping responses from the person confronted by them due to the presence of certain obstacles … [which] may

include ambiguity, uncertainty, conflicting demands, [or] lack of resources" (Nezu & D'Zurilla, 1989, p. 289). This definition of a problem includes impersonal problems … personal/intrapersonal problems … interpersonal problems, …. as well as broader community and societal problems (D'Zurilla et al., in press). A problem may be a single event, a series of related events, or a chronic situation. A solution, on the other hand, is a "situation-specific coping response or response pattern (cognitive and/or behavioral) that is the product or outcome of the problem-solving process when it is applied to a specific problematic situation (D'Zurilla et al., in press, p.3). An effective solution is one in which the coping response improves the nature of the problematic situation or reduces the emotional distress that is caused by the problematic situation (Nezu, 1987; Nezu et al., 1989). The problem-solving model proposed by Nezu and colleagues asserts that problems are variable across persons even if they experience the same type of distress because all problems represent an interaction of the individual and his or her own stressful situations (Nezu & D'Zurilla, 1989).

The social problem-solving model "was introduced by D'Zurilla and Goldfried (1971) and later expanded and refined" (D'Zurilla et al., in press). Most recently, social problem solving has been defined as consisting of "two general, partially independent processes: (1) problem orientation and (2) problem-solving style" (D'Zurilla et al., in press). Problem orientation refers to the "metacognitive process that serves an important motivational function in problem solving" whereas problem-solving style "refers to the

cognitive and behavioral activities that are aimed at finding 'solutions' or ways of coping with specific problems encountered during the course of daily living" (D'Zurilla et al., in press).

More specifically, problem orientation is "a set of beliefs, assumptions, appraisals, and expectations concerning life's problems and one's own general problem-solving ability" (Nezu et al., 1989, p.408). Problem-orientation consists of two dimensions, positive problem orientation and negative problem orientation. Positive problem orientation "is a constructive, problem-solving cognitive set that involves the general disposition to (a) appraise a problem as a 'challenge' … (b) believe that problems are solvable …, (c) believe in one's personal ability to solve problems successfully, … (d) believe that successful problem solving takes time, effort, and persistence, and (e) commit oneself to solving problems with dispatch rather than avoiding them" (D'Zurilla et al., in press). For instance, an individual with a positive problem-solving orientation believes that it is possible to quit smoking cigarettes, that he or she is capable of quitting smoking cigarettes, and is committed to taking the necessary steps toward trying to quit smoking cigarettes. "In contrast, negative problem orientation is a dysfunctional or inhibitive cognitive-emotional set that involves the general tendency to (a) view a problem as a significant threat to well-being …(b) doubt one's personal ability to solve problems successfully …, and (c) easily become frustrated and upset when confronted with problems in living" (D'Zurilla et al., in press). Thus, and individual with a negative problem orientation might report being to stressed to quit smoking, and easily give up any attempts to quit when faced with obstacles to quitting.

Problem-solving styles contain three dimensions: rational problem-solving, impulsivity/carelessness style, and avoidance style. Rational problem solving is "a constructive problem-solving style that is defined as the rational, deliberate, and systematic application of effective problem-solving skills" (D'Zurilla et al., in press). These skills include: (1) problem definition and formulation, (2) generation of alternatives, (3) decision making, and (4) solution implementation and verification (D'Zurilla et al., in press). Problem definition is defined as the "ability to understand the nature of a problem, identify obstacles to goals, delineate realistic objectives, and perceive cause-effect relationships." More specifically, a well-defined problem is one in which the individual has defined the problem, has obtained all the relevant information regarding the problem, has described the problem in clear, unambiguous language, has demonstrated minimal use of cognitive distortions about the problem or about themselves, has identified why the problem is a problem, and has set realistic goals. Generation of alternatives involves thinking "of a wide range of possible alternative ideas to solve the problem in order to maximize the likelihood that the best or most effective solution will be eventually identified" (Nezu, Nezu, Friedman, Faddis, & Houts, 1998, p.44). Decision Making involves "evaluation of each possibility and selecting the most effective" solution from the alternatives generated in the previous step, and involves a cost-benefit analysis (e.g., weighing the positive and negative short-term and long-term consequences, as well as the positive and negative consequences to the self and others). Finally solution implementation and verification involves carrying out the solution and identifying whether the solution has met the individual's goals.

According to the social problem-solving model, impulsivity/carelessness style and avoidance style are two "dysfunctional problem-solving patterns" (D'Zurilla et al., in press). Impulsivity/carelessness style is characterized by active attempts to apply problem-solving strategies and techniques, but these attempts are narrow, impulsive, careless, hurried, and incomplete" (D'Zurilla et al., in press). An individual with this style "typically considers only a few solution alternatives, often impulsively going with the first idea that comes to mind …. scans alternative solutions and consequences quickly, carelessly, and unsystematically, and monitors solution outcomes carelessly and inadequately" (D'Zurilla et al., in press). Avoidance style is "characterized by procrastination, passivity or inaction, and dependency" (D'Zurilla et al., in press). An individual characterized by this style "prefers to avoid problems rather than confronting them 'head on,' and puts off problem solving for as long as possible, waits for problems to resolve themselves, and attempts to shift the responsibility for solving his or her problems to other people" (D'Zurilla et al., in press).

Based on this model, "good social problem solvers would be characterized by high scores on measures of positive problem orientation and rational problem solving and low scores on measures of negative problem orientation, impulsivity/carelessness style, and avoidance style, whereas "poor" social problem solvers would be characterized by low scores on positive problem orientation and rational problem solving and high scores on negative problem orientation, impulsivity/carelessness style, and avoidance style" (D'Zurilla et al., in press).

The theory of social problem solving is best understood in the context of the stress model because problem solving is, essentially, a coping strategy by which individuals cope with stress (Nezu, 1987). Stress has been defined as "the reciprocal relationships among four components: 1) major negative life events, 2) daily problems, 3) negative emotional states and 4) problem-solving coping" (Nezu et al., 1998). The problem-solving model helps to explain the relationship between stressful life events, including both major negative life events as well as daily hassles, and distress, such as depression and anxiety. According to the model, "negative life events can influence depression directly as well as indirectly through their impact on the frequency of current problems, and the level of problem-solving ability" (Nezu & Ronan, 1985). Second, current problems directly and indirectly affect depression. The more effective one is at solving problems, the less distress they will experience. The more effective one is at solving problems, the fewer problems they will have. Thus, individuals who have difficulty coping with their stress will experience greater distress.

There has been extensive research evaluating the role of problem solving in explaining depression, reactions to stress, and how individuals cope with a diagnosis of cancer. Several studies have demonstrated the link between problem solving ability and depression. Specifically, problem solving has been found to serve as both a mediator and moderator for stress-related depression and anxiety (Nezu, 1985, 1986a, 1986b, 1987; Nezu, Kalmar, Ronan, & Clavijo, 1986; Nezu & Ronan, 1985). Individuals who appraise their problem-solving abilities as ineffective appeared more psychologically distressed as compared to those who appraised themselves as effective problem-solvers (Heppner,

Kampa, & Brunning, 1987). Conversely, the effective self-appraisers experience significantly less depression and anxiety. As discussed earlier, depression has been shown to correlate with smoking behavior as well as to predict relapse. Thus, an individual's ability to quit smoking may be a function of both distress as well as coping efforts to manage distress.

Over the past 15 years Nezu and colleagues have repeatedly demonstrated the stress moderating effects of problem-solving coping. In a group of 310 university students, Nezu (1986a) found that negative life stress leads to state anxiety, and problem solving moderated the effect of this stress on anxiety. Nezu (1986) and colleagues found that "individuals who have effective problem-solving skills are able to better cope with the problems and difficulties inherent in negative life stress, and, as a result, become less prone to depressive symptoms under circumstances of high stress" (Nezu et al., 1986, p. 495). A prospective study conducted by Nezu and Ronan (1988) not only supported previous findings (Nezu et al., 1986), but also found that, when prior depression was accounted for (e.g., to rule out the effects of premorbid levels of depression), problem-solving ability moderated the effects of stress on depressive symptomatology. The authors concluded that teaching clients to cope more effectively with problematic situations might serve to prevent depression when they encounter circumstances of high stress.

Actual clinical outcome studies which provide problem-solving therapy have been shown to decrease depression among people diagnosed with unipolar depression (Nezu & Perri, 1989). Recent studies have investigated the moderating role of problem solving ability and distress in people diagnosed with cancer. The Genesis Cancer Coping Project, a large-

scale, prospective outcome investigation on the effectiveness of problem-solving training for cancer patients, demonstrated problem-solving therapy to be effective in helping people with cancer improve their quality of life (Nezu, Nezu, Friedman, Houts, & Faddis, 1999). Specifically, patients who participated in problem solving therapy reported significantly lower levels of distress, had more effective problem-solving and decision-making skills, and reported significantly fewer cancer related problems as compared to individuals who received only standard medical treatment without problem-solving therapy (Nezu et al., 1999). Moreover, individuals with poorer problem-solving skills reported higher levels of depression (Nezu et al., 1999). To summarize, findings from a variety of studies support the validity of the problem solving model of stress in general (Nezu et al., 1999), as well as its applicability to persons with chronic illness, such as cancer (Nezu et al., 1999).

The present study will investigate the predictive role of problem-solving skills in smoking behavior. Why do some individuals quit smoking cigarettes upon a diagnosis, while others continue to smoke? The answer may lie in one's problem-solving skills. The problem-solving model may help explain smoking behavior among people diagnosed with either head and neck cancer or COPD. As highlighted earlier, the problem-solving model explains how individuals cope with stress, which can include major life events, daily hassles, and negative emotional states. For an individual diagnosed with head and neck cancer, the diagnosis of cancer is often, in and of itself, a major life event. Along with this diagnosis arrive a host of additional minor life events, or daily hassles, such as pain, physical disfigurement, difficulty swallowing, and speech impairment. Negative emotional states in response to a diagnosis or

treatment include depression and anger. In the case of COPD, individuals may too experience the diagnosis itself as significant. Individuals with COPD experience daily hassles such as decreased lung function, decreased sexual dysfunction due to dyspnea, and therefore, a decrease in ability to participate in hobbies, recreational activities, and other every day activities. These physical limitations often results in poorer work performance, a decrease in the quality and quantity of interpersonal relationships, and distress such as depression.

In addition to the increased number of daily hassles and the distress subsequent to a diagnosis, individuals with COPD and cancer also face the stress of quitting smoking. Ironically, the act of smoking cigarettes may be serving as their stress reducer, and such individuals may not have alternative coping resources. If, in fact, current smokers are found to have fewer problem-solving skills as compared to former smokers, then problem-solving training could provide these individuals with new coping skills. These new skills may help them to cope with their current stressors (both the major life events as well as daily hassles), thereby reducing their distress. Subsequently, the need to smoke in order to decrease their distress may decrease as well. Moreover, these new skills may help current smokers to cope with the stress of quitting. For instance, individuals may learn to more clearly identify, or define, situations in which they are likely to smoke a cigarette, or they may better generate an exhaustive list of possible solutions of how to reduce the obstacles to quitting. Ultimately, rather than being advised to quit smoking, or prescribed a medication, the individual could become an active participant in his or her care.

CHAPTER 10: THE PRESENT STUDY

Rationale

Despite the fact that a majority of individuals diagnosed with either head and neck cancer or COPD have knowledge about the negative health consequences of smoking, many continue to smoke. Although many variables have been correlated with smoking behavior and smoking cessation, few variables are unequivocally correlated, and predictors of smoking cessation are inconclusive. Research suggests a strong relationship between depression and smoking behavior such that individuals who are experiencing increased depressive symptoms under stress are more likely to be smokers. We also know that problem solving ability serves to moderate and mediate distress among individuals with a range of distress symptoms, including depression in cancer patients.

A significant amount of research in smoking behavior has been based on the Health Belief Model. The HBM has, in the past, explained underlying motivations for engaging in particular health behaviors, such as screening behaviors. An integral aspect of the model is the declaration that, among other things, an individual must perceive him- or herself to be at risk for an illness in order to perform a particular pro-health behavior. An underlying mechanism of this model is the belief in one's self-efficacy. In other words, an individual must not only believe himself to be at risk of disease, but also believe he or she can implement change (e.g., change in behavior) in order to reduce the risk. The question is: how do

individuals who are already diagnosed with cancer or COPD fit into the model? The HBM still applies to these populations. For instance, perceived susceptibility may represent susceptibility for increased symptoms or recurrence of disease in a non-primary site. Research to date has indicated self-efficacy and perceived risk to be predictors of smoking cessation in the general population.

The Transtheoretical Model, like HBM, is useful in understanding why some smokers quit successfully and others do not. While the TM is relevant to predicting which individuals successfully abstain from smoking, and in targeting and individualizing treatment for individuals characterized by earlier stages of change, like the HBM, it does not provide us with knowledge of possible interventions. Also like the HBM, the TM takes into account self-efficacy. Specifically, self-efficacy increases as one progresses through the stages of change. Utilizing these stages of change does not fit into this current study, as individuals classified as ex-smokers are, according to the TM, already past the action stage.

Although the HBM and TM explain why some smokers are successful quitters while others are not, they do not offer clinicians with solutions with which to intervene. For instance, Bandura's theory of self-efficacy, while providing an explanation for the underlying mechanisms of behavior, can only explain part of the quitting process. In addition to belief that the outcome of smoking cessation is positive, and the belief in oneself to be able to successfully quit smoking in order to reach the desired outcome, this model lacks the behavioral component of change. In other words, individuals are more likely to quit smoking if they are able to successfully cope with stressful life events such as stopping smoking.

The problem-solving model may not only offer insight into smoking behavior, but may also may provide possible solutions to the smoking cessation problem. Preliminary findings in the general, adult, nonmedical population already suggest an association between coping skills and smoking behavior such that individuals with better coping skills are more likely quit and less likely to relapse (Horowitz, 1985; Matheny & Weatherman, 1998; Shiffman et al., 1986; Wewers, 1988). If problem-solving skills predict whether an individual continues to smoke or quits smoking upon a diagnosis of either head and neck cancer or COPD, then individuals with poor problem-solving skills can be targeted for smoking cessation treatment, and furthermore, treatment exists which can help to improve an individual's problem-solving skills. Individuals with good problem-solving skills may be better able to cope with and resolve problems in life, such as daily hassles, and overall, to cope better with stress. Individuals with good problem-solving skills may better cope with the specific problems and stress inherent in attempting to quit smoking, such as coping with withdrawal symptoms, increased distress, and exposure to smoking triggers and cues.

The present study will examine, among other variables, the predictive value of problem solving on smoking behavior. This will be the first time problem solving will be examined in its relation to actual smoking behavior change. The purpose of studying two distinct, chronically ill populations is two-fold. First, the study will investigate predictors of smoking cessation in each population to better understand how clinicians might identify patients who require additional skills or therapy in order to quit successfully. However, the smoking cessation rates between these two populations are significantly different, with

continued abstinence significantly greater among individuals with head and neck cancer. Therefore, if these different rates occur in this sample, we may compare the two populations, investigate differences, and apply the information gleaned to help all individuals in both groups to reduce smoking and improve health.

Original Research Questions and Hypotheses

Question 1: Are the cessation rates for cancer and for COPD significantly different?

Hypothesis 1: Based on previous literature, cessation rates will be significantly different between the two populations.

Question 2: Does medical severity predict smoking status above and beyond demographic variables?

Hypothesis 2: After controlling for demographic variables, medical severity will predict whether a participant is a current smoker or former smoker.

Question 3: Do smoking history variables predict smoking status above and beyond demographic variables and medical severity?

Hypothesis 3: After controlling for demographic variables and medical severity, smoking history variables (e.g., pack years and nicotine dependence) will predict smoking status.

Question 4: Does alcohol use predict smoking status above and beyond demographic, medical severity, and smoking history variables?

Hypothesis 4: Alcohol use (e.g., number of alcoholic beverages per week) will predict smoking status above and beyond demographic, medical severity, and smoking history variables.

Question 5: Does mood predict whether a participant is a smoker or former smoker?

Hypothesis 5: After controlling for demographic variables, medical severity, smoking history variables, and alcohol variables, depressive and anger symptoms will predict the probability of being a current smoker or former smoker.

Question 6: Does problem solving predict whether a participant is a current smoker or former smoker?

Hypothesis 6: After controlling for demographic variables, medical severity, smoking history variables, alcohol variables, and mood variables, problem-solving ability will predict the probability of being a current smoker or former smoker.

Rationale for Revising Research Questions and Hypotheses

Due to the small sample size of each group, there was not enough power to conduct two separate logistic regressions. Therefore, the two medical populations were combined in order to increase the sample size, and therefore the power, of the logistic regression analysis. The prognostic indicator (i.e., severity ratings) for the two populations differ, and cannot be collapsed into one measure of severity for both COPD and head and neck cancer. Therefore, medical severity was eliminated as a predictor variable. Limitations regarding this elimination will be discussed in the Limitations section. However, diagnosis as a dichotomous variable was added as a predictor into the logistic regression analysis so as to account for any differences between current smokers and former smokers due to medical diagnosis.

Revised Research Questions and Hypotheses

Research questions # 1 remains the same.

Question 1: Are the cessation rates for cancer and for COPD significantly different?

Hypothesis 1: Based on previous literature, cessation rates will be significantly different between the two populations.

Research question #2 changes from medical severity to medical diagnosis.

Question 2: Does medical diagnosis predict smoking status above and beyond demographic variables?

Hypothesis 2: After controlling for demographic variables, medical severity will predict whether a participant is a current smoker or former smoker.

Research questions #3 through #6 are essentially the same except that we are controlling for medical diagnosis rather than medical severity.

Question 3: Do smoking history variables predict smoking status above and beyond demographic variables and diagnosis?

Hypothesis 3: After controlling for demographic variables and diagnosis, smoking history variables (e.g., pack years and nicotine dependence) will predict smoking status.

Question 4: Does alcohol use predict smoking status above and beyond demographic, diagnostic, and smoking history variables?

Hypothesis 4: Alcohol use (e.g., number of alcoholic beverages per week) will predict smoking status above and beyond demographic, diagnosis, and smoking history variables.

Question 5: Does mood predict whether a participant is a smoker or former smoker?

Hypothesis 5: After controlling for demographic variables, diagnosis, smoking history variables, and alcohol variables, depressive and anger symptoms will predict the probability of being a current smoker or former smoker.

Question 6: Does problem solving predict whether a participant is a current smoker or former smoker?

Hypothesis 6: After controlling for demographic variables, diagnosis, smoking history variables, alcohol variables, and mood variables, problem-solving ability will predict the probability of being a current smoker or former smoker.

CHAPTER 11: MATERIALS AND METHODS

Participants

Participants with COPD were recruited from two urban, outpatient clinics, at MCP Hahnemann University and Temple University Hospital Lung Center. Participants with head and neck cancer were recruited from two, urban, outpatient physician's offices, also at MCP Hahnemann University and Temple University Hospital. Inclusion criteria included: 1) a diagnosis of either COPD or head and neck cancer, 2) existence of a smoking history, past or present, and 3) between the ages of 25 to 80. This age range was chosen based on ages accepted in prior studies investigating head and neck cancer and COPD. Furthermore, although the average age in prior studies has been approximately 50 years of age, the range has varied. Cancer studies show up to 25% of the population being greater than or equal to 70 years of age. COPD studies have accepted individuals up to 80 years of age. Exclusion criteria included any known history of mental illness including bipolar disorder, psychosis and/or mental retardation.

One-hundred and three out of the 624 patients approached participated in this study. Reasons for not participating in the study are listed in Table 2.

One-hundred and three participants completed and returned all questionnaires. Three follow-up attempts were made for each participant. The 76 participants who completed the follow-up were classified into the same smoking category as they were

classified at initial participation. Twenty-seven of the 103 participants could not be reached for the 3-month follow-up, and were therefore classified into the same smoking status in which they had been classified upon their initial participation. More specifically, 8 had telephone numbers which were disconnected, 14 did not answer their telephone, 4 were deceased, and 1 refused to participate in the follow-up assessment. Ten of these individuals were classified as current smokers at initial participation. Their smoking rate ranged from 1 cigarette per week to 10 cigarettes per day. Seventeen of the individuals were classified as former smokers at initial participation, and the amount of time since quit date ranged from 1 month to 22 years. Furthermore, 4 of these individuals had quit within the past year. Ultimately, because not every participant was reached for their 3-month follow-up, reliability of classification is diminished. It is possible that the individuals who were not reached might have changed their smoking habits (i.e., current smokers at initial participation might have quit smoking and former smokers at initial participation might have picked up smoking).

Table 2. Reasons for not participating

Reason	Head and Neck Cancer	COPD	Combined
Declined	43	3	46
Never smoked	60	1	61
Quit before diagnosis	70	13	83
Younger than 25 y.o.	7	4	11
Older than 80 y.o.	35	4	39
Not diagnosed with cancer	207	N/A	207
Not diagnosed with COPD	N/A	58	58
Non-English speaking	5	1	6
Non-smoking-related malignancy	9	N/A	9
Post lung transplant	N/A	1	1

Materials

Demographic and smoking history information.

The demographic questionnaire (See Appendix A) was developed and based on a demographic questionnaire used in the Genesis Cancer Coping Project. Demographic information was obtained using a semi-structured interview.

Smoking history variables are found in the Demographic Questionnaire, and were also obtained using a semi-structured interview. The smoking history variables include measures that have been found, at least in some studies, to predict smoking cessation in the past. Smoking variables that were examined and entered into the logistic regression include 1) pack years and 2) a measure of nicotine dependence ("how soon after you wake up do you smoke your first cigarette"). Additional information regarding the participants' smoking habits was explored for informational purposes only, and not for inclusion in the regression equation. Some of the smoking habit questions were adapted from the Mayo Foundation Nicotine Dependence Center Patient Questionnaire (Mayo Foundation, 1993), while others were adapted from previous research. For instance, nicotine, which is contained in all tobacco products, is considered an addictive substance, and therefore, common withdrawal symptoms which have been well documented, were assessed, including craving, difficulty concentrating, increased appetite, insomnia, irritability, restlessness, and decreased heart rate (Greden & Pomerlau, 1995; Lillington, Leonard, & Sachs, 2000). While degree of nicotine

is not being assessed per se, one variable which is assessed here, and which has been shown to correlate with nicotine dependency, is time to first cigarette after awakening in the morning (Gritz et al., 1993; Lillington et al., 2000,

p. 202).

A measure of alcohol use was assessed based on a study conducted by Murray et al. (1995), which asked the following two questions: 1) how many days per week, on average, do you drink alcoholic beverages, and 2) on the days you drink alcohol, how many drinks, on average, do you have? Number of drinks per day multiplied by number of days per week was computed to provide the average number of drinks consumed per week.

Risk perception was measured by the question "Do you believe quitting smoking would be beneficial to your health" (for current smokers) and "Do you think quitting smoking was beneficial to your health" (for former smokers). Previous studies investigating knowledge of risk have also used single items to assess this variable (Brownson et al., 1992; Lee, 1989; Strecher et al., 1995).

Social problem solving information.

Problem solving was measured using the Social Problem-Solving Inventory - Revised (SPSI-R; D'Zurilla, Nezu & Maydeu-Olivares, 2002). The SPSI-R is a 52-item self-report measuring social problem-solving ability based on several problem-solving components: problem orientation, problem definition and formulation, generation of

alternatives, decision-making, and solution implementation and verification (see Appendix B). The five scales which reflect these components are Negative Problem Orientation, Positive Problem Orientation, Rational Problem Solving, Impulsivity/Carelessness Style and Avoidance Style. Each item on the scale depicts a positive or negative cognitive, affective, or behavioral response to a problem situation and is rated by the patient from 0 (not at all true of me) to 4 (extremely true of me). Negative Problem Orientation is "a general set that influences a person to view problems as threats, expect problems to be unsolvable, [and to] doubt one's own ability to solve problems successfully" (Nezu, Nezu, Houts, Friedman, & Faddis, 1999, p. 16). Positive Problem Orientation is "a general set that leads a person to appraise problems as challenges, have the optimistic belief that problems are solvable [and] perceive one's own ability to solve problems as high" (Nezu, Nezu, Houts, Friedman, & Faddis, 1999, p. 16). Rational Problem-Solving is a "rational, systematic, and skillful application of various effective problem-solving strategies"(Nezu, Nezu, Houts, Friedman, & Faddis, 1999, p. 16) such as components of problem definition, generation of alternatives and decision making. Impulsivity/Carelessness Style is "a generalized problem-solving style characterized by impulsive, hurried, and careless attempts to solve problems" (Nezu, Nezu, Houts, Friedman, & Faddis, 1999, p. 16). Avoidance Style is "a second maladaptive problem-solving style characterized by procrastination, passivity, and dependency" (Nezu, Nezu, Houts, Friedman, & Faddis, 1999, p.16). The measure is found to have good test-retest reliability for all five scales and the total scale, with Cronbach's alpha ranging from .72 to .91, good internal consistency, with Cronbach's alpha ranging from .69 - .95, and good

concurrent validity as compared with the Means-Ends Problem-Solving Procedure (D'Zurilla & Nezu, 1990).

Mood information.

Depression and anger were measured using the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1992). The POMS (See Appendix C) is a 65-item self-report in which individuals are asked to indicate how various feelings describe them over the past week, ranging from 0 (not at all) to 4 (extremely). The POMS is comprised of six categories of mood states, including Anger-Hostility, Confusion-Bewilderment, Depression-Dejection, Fatigue-Inertia, Tension-Anxiety, and Vigor-Activity. The two scales which were analyzed in the present study were the Depression-Dejection and Anger-Hostility subscales, which contain 15 and 12 items, respectively. The POMS has an internal consistency of .95 in two separate studies, and has been used in a wide variety of research studies (Nezu, Nezu, McClure, & Zwick, 2002). The POMS is a widely used measure of negative affect in smoking cessation studies (Gilbert et al., 1997).

Patient diagnostic information.

Medical information such as diagnosis, disease severity, and treatment, were obtained from each patient's medical chart and recorded in the Patient Diagnostic Form (see Appendix D).

Procedure

Participants were recruited from the physician's waiting room by either the author of this study or by a doctoral candidate in clinical psychology. The doctoral candidate worked in the same research lab that the author had worked in, was familiar with the SPSI-R, and had collected data for other studies, including clinical outcome studies. In addition, the author trained the doctoral candidate in data collection procedures particular to this study and reviewed all measures, including all questions on the Demographic Form, in order to increase reliability and to reduce stylistic differences. First, the doctoral candidate observed the author accrue participants and collect data. Second, the author observed the doctoral candidate accrue participants and collect data. An administrator at each site provided the data collector with a list of individuals who met medical diagnostic criteria. The data collector approached potential participants, described the study, and asked the person to volunteer. Persons interested in volunteering read and signed the Informed Consent (see Appendix E and Appendix F). Individuals who signed the Informed Consent then participated in a semi-structured interview (the Demographic Form) and completed two self-reports (the SPSI-R

and the POMS). The semi-structured interview and completion of the self-reports took place in a private room at the physician's office. Participants were encouraged to complete the interview and self-report measures while waiting for their appointment. In order to ensure anonymity and confidentiality, all participants were assigned a number, known only to the PI, and only this number appeared on the questionnaires. In addition, confidentiality of responses was verbally emphasized immediately prior to the interview. Medical information such as diagnosis, disease severity, and treatment, were obtained from the participants' medical charts and recorded in the Patient Diagnostic Form. Finally, a three-month follow-up was conducted. Participants were contacted by telephone three months after their initial participation and asked about their smoking status and habits. This information was recorded on the Three-Month Follow-Up Form (see Appendix F).

CHAPTER 12: ANALYTIC APPROACH

The overall goal of this research was to investigate predictors of smoking behavior in people with head and neck cancer or COPD. Research to date suggests that demographic, smoking, and alcohol variables predict smoking behavior (e.g., smoking cessation) in the general population. This study looks at 1) whether these variables predict smoking behavior upon a diagnosis of either head and neck cancer or COPD, 2) whether mood (e.g., depression, anger) can predict smoking behavior above and beyond demographic, smoking and alcohol variables in these two populations, and 3) whether problem-solving skills can predict smoking behavior above and beyond the aforementioned variables in these two populations.

To address these research questions, a logistic regression model was developed to investigate potential demographic, diagnostic, smoking history, alcohol use, and psychosocial predictors of continued smoking in patients with head and neck cancer and COPD. First, bivariate relationships were tested between all independent (predictor) variables and measures of smoking status. The bivariate correlations are presented in Table 3. Next, the set of predictors were included in a hierarchical logistic regression, with stepwise method of variable entry within the demographic variables block, which contained multiple variables. Each block represented a group of related variables. The blocks were entered in the following order: demographic variables, diagnostic variable, smoking variables, alcohol variable, mood variables, and problem-solving variable. Any variable which had too much

missing data or lacked variability in distribution was dropped from the analysis. A list of variables investigated is presented in Table 4.

Table 3. Pearson correlations between predictor variables and outcome variable

Pearson Correlations	Smoking Status
Gender	-.003
Age	.002
Educational attainment	-.015
Employment status	-.004
Married or living with a significant other	-.009
Single	.248*
Separated, divorce, or widowed	.233*
Employed	.065
Retired	-.110
Disability	.056
Ethnicity (white/non-white)	.078
Diagnosis	-.072
Pack years	.082
Nicotine dependence	.138
Age smoked first cigarette	.030
Has tried to cut down smoking	-.208*
Longest time ever stopped smoking	-.573**
Alcohol use	. 235*
POMS Depression	-.071
POMS Anger	.037
SPSI Total	-.014

* Correlation is significant at the 0.05 level (2-tailed). ** Correlation is significant at the 0.01 level.

Table 4. List of predictor variables examined

Category	Variable
Demographic	Age Gender Ethnicity Employment status Marital status Years of education Income [a]
Diagnostic	COPD or head and neck cancer
Smoking variables	Pack years [b] Nicotine dependence [c]
Alcohol use	Number of alcoholic beverages consumed per week
Mood	POMS depression/dejection scale POMS anger/hostility scale
Problem solving	SPSI-R total score

[a] excluded from logistic regression analysis due to missing data. [b] pack years equals number of packs smoked per day times number of years smoked. [c] nicotine dependence was measured by the question "how soon after you wake up do you smoke your first cigarette?" This variable was entered as a dichotomous variable (e.g., < 30 min and 30 min or greater.

CHAPTER 13: RESULTS

Descriptive Data According to Medical Diagnosis

Demographics.

Fifty-four patients with COPD and 49 patients with head and neck cancer participated in the study, resulting in a total of 103 participants. The sample was evenly divided between males and females. 58.3% were Caucasian, 39.8% were African American, and 1.9% were Hispanic. Over one half of the participants were either married or living with a significant other. Seventy-four percent of the sample were either retired or on disability. Overall, the COPD and head and neck cancer groups did not significantly differ on highest degree attained, gender, or marital status. There was a significant difference between the COPD and head and neck cancer groups on age ($t(101) = 2.239$; $p=.027$), ethnicity ($\chi^2 (1) = 11.446$; $p=.001$) and income ($\chi^2 (4) = 13.902$, $p=.008$). Individuals with COPD tended to be older. A majority of the head and neck cancer population tended to be Caucasian, whereas the COPD population was more evenly distributed between Caucasians and African-Americans. Data on these demographic variables is presented in Tables 5 and 6.

Table 5. Means, standard deviations and medians for demographic data according to diagnosis

Variable	COPD		Head and Neck Cancer	
	M (SD)	*Mdn*	*M (SD)*	*Mdn*
Age [a]	54 (9.52)	64.0	49 (9.11)	58.0
Average number of years of education	12.19 (2.79)	12.0	12.94 (2.61)	12.0

[a] significant difference between groups

Table 6. Frequencies and percents for demographic data according to diagnosis

Variable	COPD		HNC	
	Frequency	Percent	Frequency	Percent
Ethnicity [a]				
African-American	30	55.6	11	22.4
Caucasian	23	42.6	37	78.5
Hispanic	1	1.9	1	2.0
Gender				
Female	30	63	14	28.6
Male	24	44.4	29	59.2
Income [a]				
$0 - $24,000	34	63	14	28.6
$25,000 - $49,999	13	24.1	9	18.4
$50,000 - $74,000	2	3.7	8	16.3
$75,000 - $99,000	0	0	3	6.1
$100,000 and up	5	9.3	2	4.1
Refused to disclose	1	1.9	13	26.5
Living status				
Single	6	11.1	5	10.2
Living with sig. Other	1	1.9	3	6.1
Married	22	40.7	28	57.1
Separated	2	3.7	2	4.1
Divorced	10	18.5	8	16.3
Widowed	13	24.1	3	6.1
Employment status				
Employed	11	20.4	17	34.7
Retired	24	44.4	20	40.8
Disability	19	44.2	12	24.5

[a] significant difference between groups

Medical variables.

A majority of the participants with head and neck cancer had cancer of the oral cavity, pharynx, or larynx (see Table 7). The severity of illness at which people with head and neck cancer were diagnosed was approximately equal across TNM stages (see Table 8). Over 90% of patients had undergone surgery and over 70% had received radiation treatment.

Table 7. Participants with head and neck cancer broken down by primary site

Cancer Diagnosis	N	Percent
Oral Cavity	17	34.7
Pharynx	15	30.6
Larynx	9	18.4
Maxillary Sinus	2	4.1
Salivary Glands	3	6.1
Larynx and Pharynx	2	4.1
Unknown Primary	1	2.0

Table 8. Participants with head and neck cancer broken down by TNM stage at diagnosis

TNM Stage	N	Percent
Stage I	11	22.4
Stage II	10	20.4
Stage III	12	24.5
Stage IV	13	26.5
Not in medical chart	3	6.1

Among the participants with COPD, severity of illness was evenly distributed (see Table 9). Individuals with COPD took an average of 4 medications per day to specifically treat their pulmonary disease.

Table 9. Participants with COPD broken down by FEV1% predicted

FEV1% Predicted	N	Percent
50 – 70	18	33.0
35-49	20	37.0
< 35	16	29.9

Smoking variables.

The majority of participants in each diagnostic group had stopped smoking. In this sample, 31.5% of COPD participants and 26.5% of participants with head and neck cancer continue to smoke cigarettes, with the remaining in each group consisting of former smokers (see Table 10).

Table 10. Smoking status according to diagnosis

Smoking Status	COPD	Head and Neck Cancer	Total
	Frequency (Percent)	Frequency (Percent)	Frequency (Percent)
Current Smoker	17 (31.5)	13 (26.5)	30 (29.1)
Former Smoker	37 (68.5)	36 (73.5)	73 (70.9)
Total	54 (100.0)	49 (100.0)	103 (100.0)

While there were no significant differences between the COPD and head and neck cancer groups along a majority of smoking variables, several variables did yield significant differences. Individuals with COPD were more likely than individuals with head and neck cancer to smoke in the presence of their children (χ^2 (1) = 4.255, df=1; p=.039). Significantly more individuals with COPD than head and neck cancer reported experiencing withdrawal symptoms such as craving (χ^2 (2) 6.348; p=.042) and increased eating (χ^2 (4) 12.384; p=.015). More individuals with COPD than head and neck cancer reported having tried to cut down their cigarette consumption in the past (χ^2 (1) 4.669; p=.031), or had attempted to stop smoking (χ^2 (2) 5.616; p=.060). In addition, significantly more individuals in the COPD group had tried various quit methods such as support groups (χ^2 (1)= 3.376; p=.066), nicotine gum (χ^2 (1) 3.073; p=.080), and bupropion (χ^2 (1) 10.619; p=.001). Means, standard deviations, and medians for smoking variables are presented in Table 11. Frequencies and percents for smoking variables are presented in Table 12.

Table 11. Means, standard deviations, and medians for smoking variables by diagnosis

Variable	COPD		HNC	
	M (SD)	*Mdn*	*M (SD)*	*Mdn*
Age of first cigarette	14.3 (3.7)	14.5	14.6 (4.3)	14.0
Age of regular daily cigarette smoking	17.0 (3.8)	16.0	18.2 (5.5)	18.0
Average number of cpd at diagnosis	26.1 (14.2)	20.0	24.7 (13.6)	20.0
Average number of cpd, current	4.09 (8.9)	.00	2.6 (7.4)	.00
Average number of cpd, past 6 months	4.24 (8.82)	.00	2.84 (7.59)	.00
Average cpd when smoking was the heaviest	34.3 (15.4)	30.0	34.0 (14.7)	30.0
Average cpd over lifetime	25.2 (11.5)	20.0	23.7 (11.1)	20.0
Average cpd at time of quit	20.6 (13.3)		21.1 (12.5)	
Pack years	53.1 (27.6)	46.5	46.6 (31.0)	37.0
Number times stopped smoking for at least 1 day	8.9 (16.5)	3.0	5.2 (7.9)	2.5
Number of alcoholic beverages/week	1.70 (3.56)	.00	4.80 (9.15)	.00
Number of other smokers in the household	.5 (.6)	.00	.8 (2.9)	.00

Note. cpd = cigarettes per day.

Table 12. Frequencies and percents of smoking variables according to diagnosis

Variable	COPD		HNC	
	Frequency	Percent	Frequency	Percent
Participants that have attempted to cut down smoking	52	96.3	41	83.7
Participants that have attempted to stop smoking	53	98.1	43	87.8
Participants who experienced withdrawal symptoms				
Anxiety	25	46.3	21	42.9
Craving*	37	68.5	25	51.0
Restlessness	24	44.4	21	42.9
Increased eating	35	64.8	19	38.8
Difficulty concentrating	10	18.5	11	22.4
Increased irritability	25	46.3	21	42.9
Participants who have used other tobacco products				
Snuff	0	0	0	0
Chewing tobacco	0	0	1	2.0
Cigars	8	14.8	12	24.5

Pipe	9	16.7	11	22.4

Table 12 (continued)

Variable	COPD		HNC	
	Frequency	Percent	Frequency	Percent
Participants who used the following quit methods				
"Cold turkey"	43	79.6	44	89.8
Support group*	8	14.8	2	4.1
Nicotine gum*	14	25.9	6	12.2
Bupropion*	13	24.1	1	2.0
Nicotine patch	20	37.0	8	16.3
Cigarette participants found most difficult to give up:				
First cigarette of the Morning	20	37.0	12	24.5
After meals	14	25.9	13	26.5
During or after stressful situations	11	20.4	12	24.5
During social occasions	8	14.8	9	18.4
Could not choose	1	1.9	3	6.1
How soon participants smoke upon awakening:				
Within 30 minutes	41	75.9	35	71.4
Greater than 30 Minutes	13	24.1	14	26.6

* $p < .05$

Psychosocial variables.

There were no significant differences between the COPD and head and neck cancer groups on measures of anger or problem-solving skills. There was also no significant difference between the groups along the subscale measures of problem solving. However, individuals in the COPD group reported more symptoms of depression according to the POMS as compared to individuals in the head and neck cancer group ($t(101) = 3.099$; $p=.002$). Means, standard deviations, and medians for these variables are reported in Table 13.

Table 13. Means, standard deviations, and medians for psychosocial variables by diagnosis

Variable	COPD		HNC	
	M (SD)	*Mdn*	*M (SD)*	*Mdn*
POMS Anger	8.3 (1.2)	5.5	6.2 (6.3)	5.0
POMS Depression	13.0 (11.9)	11.0	8.7 (9.0)	5.0
POMS Total	40.6 (39.6)	35.0	19.1 (29.3)	16.0
SPSI Total	13.7 (3.2)	14.0	13.9 (3.2)	14.4

Descriptive Data According to Smoking Status

Demographic variables.

There was a total of 30 current smokers and 73 former smokers across both medical populations. There were no significant differences between the groups on age, ethnicity, gender, income or occupation. The difference between groups on educational attainment approached significance ($\chi 2$ (5) = 9.654; p=.086). There was a significant differences between groups on marital status ($\chi 2$ = 7.109; p=.029). Former smokers tended to be married whereas current smokers tended to be separated, divorced or widowed. Moreover, individuals who were separated, divorced, or widowed were approximately 5 times as likely to be a current smoker as compared to individuals who were married or living with a significant other.

Medical variables.

As noted earlier, 31.5% of COPD and 26.5% of head and neck cancer continue to smoke cigarettes, with the remaining in each group consisting of former smokers. No other medical variables, such as medical severity, could be utilized to directly compare groups because disease severity and types of treatment are different in these two populations.

Smoking variables.

As expected, there was a significant difference between groups on current smoking status ($t(29) = 5.372$; $p=.000$). Means, Standard deviations, and medians for smoking variables are presented in Table 14. Frequency and percents are presented in Table 15.

Table 14. Means, standard deviations, and medians for smoking history variables according to smoking status

Variable	Current Smoker		Former Smoker	
	M (SD)	*Mdn*	*M (SD)*	*Mdn*
Age of first cigarette	14.57 (3.47)	15.00	14.40 (4.13)	14.00
Age of regular daily cigarette smoking	17.60 (4.34)	16.00	17.58 (4.89)	17.00
Average number of cpd at diagnosis	26.80 (13.55)	25.00	24.88 (13.99)	20.00
Average number of cpd, current	11.57 (11.79)	8.00	---	---
Average number of cpd, past 6 months	12.27 (11.36)	10.00	---	---
Average cpd when smoking was the heaviest	36.00 (14.35)	35.00	33.38 (15.31)	30.00
Average cpd over lifetime	24.90 (10.17)	20.00	24.34 (11.81)	20.00
Average cpd at time of quit	---	---	20.88 (12.86)	20.00
Pack years	54.11 (25.49)	46.00	48.27 (30.73)	43.00
Number times stopped smoking for at least 1 day	9.28 (19.77)	4.00	6.24 (9.27)	2.00

Table 14 (continued)

Variable	Current Smoker		Former Smoker	
	M (SD)	*Mdn*	*M (SD)*	*Mdn*
Number of alcoholic beverages/week	5.60 (10.75)	.00	2.18	.00
Number of other smokers in the household	1.03 (3.62)	.00	.48 (.69)	.00

Table 15. Frequencies and percents for smoking history variables according to smoking status

Variable	Current Smoker		Former Smoker	
	Frequency	Percent	Frequency	Percent
Participants that have attempted to cut down smoking in the past	30	100	63	93.2
Participants that have attempted to stop smoking in the past	28	93.3	68	93.2
Participants who experienced withdrawal symptoms				
Anxiety	14	46.7	32	43.8
Craving*	16	53.3	46	63.0
Restlessness	15	50.0	30	41.1
Increased eating	13	43.3	41	56.2
Difficulty concentrating	5	16.7	6	8.2
Increased irritability	17	56.7	29	39.7
Participants who have used other tobacco products				
Snuff	0	0	0	0
Chewing tobacco	0	0	1	1.4
Cigars	10	33.3	11	15.1

Pipe	8	26.7	12	16.4

Table 15 (continued)

Variable	Current Smoker		Former Smoker	
	Frequency	Percent	Frequency	Percent
Participants who used the following quit methods				
"Cold turkey"	21	70	66	90.4
Support group*	4	13.3	6	8.2
Nicotine gum*	8	26.7	12	16.4
Bupropion*	6	20.0	8	11.0
Nicotine patch	9	30.0	19	26.0
Cigarette participants found most difficult to give up:				
First cigarette of the morning	7	23.3	25	34.2
After meals	8	26.7	19	26.0
During or after stressful situations	9	30.0	14	19.2
During social occasions	6	20.0	11	15.1
Could not choose	0	.00	4	5.5
How soon participants smoke upon awakening:				

Within 30 minutes	20	66.7	56	76.7
Greater than 30 Minutes	10	33.3	17	23.3

Table 15 (continued)

Variable	Current Smoker		Former Smoker	
	Frequency	Percent	Frequency	Percent
When do you/did you smoke the heaviest				
Mornings	10	33.3	8	11.0
Afternoons	2	6.7	13	17.8
Evenings	14	46.7	37	50.7
Throughout the day	4	5.5	15	20.5

Significantly more former smokers than current smokers had tried to cut down or limit their smoking in the past ($\chi 2(1) = 4.551$; p=.033). Significantly more former smokers reported smoking in their car when non-smokers were with them ($\chi 2(1) = 8.320$; p=.004) and were more likely to smoke in other peoples' cars ($\chi 2(1) = 3.959$; p=.047). Significantly more former smokers had smoked in the presence of their children than current smokers ($\chi 2(1) = 5.652$; p=.017). In terms of quitting techniques, significantly more former smokers had attempted to quit "cold turkey" ($\chi 2(1) = 6.751$; p=.009). Otherwise, there were no significant differences between these groups on quitting variables. There were no significant differences with respect to withdrawal symptoms. There was a significant difference between groups in terms of when former smokers and current smokers smoked the heaviest ($\chi 2(3) = 8.572$; p=.036). More specifically, former smokers tended to have smoked the heaviest in the evenings. There was a significant difference between the two groups based on the longest ever stopped smoking ($\chi 2(4) = 34.796$; p=.000) whereby a majority of former smokers had a quit attempt greater than one year. Former smokers were more likely to have smoked in other people's homes ($\chi 2(1) = 5.988$; p=.014) as well as in public ($\chi 2(1) = 8.842$; p=.003), in restaurants ($\chi 2(1) = 23.499$; p=.000), and at work ($\chi 2(1) = 5.411$; p=.020). Most former smokers had smoked in public and in restaurants, whereas approximately half of current smokers smoke in restaurants.

Psychosocial variables.

There were no significant differences between former smokers and current smokers on measures of depression, anger, and problem solving skills. Means, standard deviations, and medians for psychosocial variables according to smoking status are presented in Table 16.

Table 16. Means, standard deviations, and medians for psychosocial variables according to smoking status

Variable	Current Smoker		Former Smoker	
	M (SD)	*Mdn*	*M (SD)*	*Mdn*
POMS Anger	7.83 (8.09)	9.50	7.10 (7.57)	5.00
POMS Depression	10.63 (10.92)	9.20	11.10 (10.81)	9.00
POMS Total	30.80 (38.35)	18.50	30.22 (36.03)	24.00
SPSI Total	13.43 (2.78)	14.02	13.76 (3.00)	14.42

Logistic Regression

The results of the logistic regression are presented in Table 17. In the first step, demographic variables were entered. Using a stepwise method of entry, only marital status was significant and entered into the equation. Marital status significantly contributed to the equation, and accounted for 9.9% of the variance in smoking status. Demographic variables, as a whole, predicted smoking status.

In the second step, diagnosis was the only variable entered. Diagnosis did not significantly add to the prediction of smoking status above and beyond demographic variables, and the overall model remained nonsignificant. Diagnosis did not raise the amount of variance accounted for in smoking status.

In the third step, smoking variables were entered. Pack years and time to first cigarette in the morning (a measure of nicotine dependence) were entered into the equation. These variables did not significantly add to the prediction of smoking status, and the overall model remained nonsignificant. The smoking history variables raised the amount of variance accounted for to 12.3%.

In the fourth step, the alcohol variable (number of alcoholic beverages consumed per week) was entered. Alcohol significantly contributed to the equation and increased the variance accounted for to 19.5%.

In the fifth step, both mood variables were entered. Neither depression nor anger were significant. The overall model remained significant, with 22.9% of the variance accounted for.

In the sixth and final step, problem solving was entered. This variable was not significant. The overall model remained significant. However, total variance accounted for did not increase, and remained at 22.9%.

Table 17. Variables entered in the stepwise logistic regression analysis for smoking status

Variable	Step χ^2	Df	Sig.	Model χ^2	Df	Sig.	Nagelkerke R^2
Demographics	7.38	2	.03*	7.38	2	.03*	.099
Diagnosis	.001	1	.97	7.38	3	.06	.099
Smoking history	1.92	2	.38	9.30	5	.10	.123
Alcohol	5.86	1	.02*	15.16	6	.02*	.195
Mood	2.85	2	.24	18.01	8	.02*	.229
Problem solving	.019	1	.89	18.03	9	.03*	.229

* $p < .05$

Next, independent variables that were specifically predictive of smoking status were examined. A list of variables included in the equation are presented in Table 18. In the final equation, only marital status and alcohol use were significant.

Table 18. Variables in the equation

Variable	B	S.E.	Wald	df	Sig.	R	Exp(B)	95.0% C.I. for Exp(B)	
								Lower	Upper
Marital Status *a*			8.466	2	.015	.205			
Marital *b*	-.004	.999	.0000	1	.996	.000	.996	.140	7.061
Marital *c*	1.607	.569	7.986	1	.005	.237	4.987	1.636	15.198
Diagnosis	-.242	.518	.217	1	.641	.000	.786	.285	2.168
Pack years	.009	.009	1.002	1	.317	.000	1.009	.992	1.026
Time to first cigarette in the morning	-.358	.562	.405	1	.524	.000	.699	.232	2.105
ETOH	.085	.042	4.162	1	.041	.143	1.088	1.003	1.180
POMS Depr	-.059	.034	2.089	1	.148	-.029	.952	.890	1.018
POMS Ang	.0680	.045	2.281	1	.131	.051	1.070	.980	1.169
SPSI Total	.0128	.093	.019	1	.891	.000	1.013	.844	1.215
Constant	-1.850	1.929	.920	1	.338				

a married or living with significant other. *b* single. *c* separated, divorced, or widowed
$p < .05$

This model is only moderately predictive of smoking status (R^2 = 22.9%). Although the model correctly classifies 78% of the cases (see Table 19), this is only slightly higher than the base rate classification of 72% (i.e., if nothing was known about the patient, he or she would be classified into the larger category of former smokers).

Table 19. Classification table for model

Observed	Predicted		Percentage Correct
	Former Smoker	Current Smoker	
Former Smoker	70	3	95.89
Current Smoker	19	11	36.67
Overall Percentage			78.64

Note. The cut value is .500

Multicollinearity

Independent variables were investigated for multicollinearity. The only two predictor variables which were found to highly correlate were POMS depression and POMS anger (r (n-1) = .89). This correlation was expected, and neither variable was removed from the analysis because both variables are directly related to the hypotheses.

Residual Analysis

A residual analysis was conducted on the logistic model in order to identify potential outliers or unusual data points (i.e., cases) in this sample. Using several measures, including Cook's distance, Leverage point, standardized residual (Z-score), and DFBETA, one case was high on more than one indicator, and was found to have significant influence on the regression equation. Briefly, the standardized residual identifies outliers that are not represented by the regression equation; an "observation is termed a leverage point if it has substantial impact on the regression results due to its differences from other observations on one or more of the independent variables (Hair, Anderson, Tatham, & Black, 1995, p. 152); DFBETA is a "measure of the change in a regression coefficient when an observation is omitted from the regression analysis" (Hair et al., 1995, p. 151), and Cook's distance is "a measure of the influence of a single case (observation) based on the total changes in all other residuals when the case is deleted from the estimation process" (Hair et al., 1995, p. 151).

This case had a standardized residual greater than 2.58, a leverage value out of range, a small DFBETA, Cook's distance greater than 1, and high leverage point. When this case was reviewed, variable by variable, the case was an outlier on two important measures, the POMS depression and the POMS anger. When this case was removed from the logistic regression analysis, the results of the analysis changed.

Logistic Regression with Outlier Removed

The results of the logistic regression when the outlier was removed are presented in Table 20.

In the first step, demographic variables were entered. Similar to the initial analysis, only marital status was significant and entered into the equation. Marital status significantly contributed to the equation, and accounted for 8.9% of the variance in smoking status. Demographic variables, as a whole, did not predict smoking status.

In the second step, diagnosis was entered. Similar to the initial analysis, diagnosis did not significantly add to the prediction of smoking status above and beyond demographic variables, and the overall model remained nonsignificant. Diagnosis did not raise the amount of variance accounted for in smoking status.

In the third step, smoking variables were entered. Pack years and time to first cigarette in the morning (a measure of nicotine dependence) were entered into the equation. Similar to the initial model, these variables did not significantly add to the prediction of

smoking status, and the overall model remained nonsignificant. The smoking history variables raised the amount of variance accounted for to 10.8%.

In the fourth step, the alcohol variable was entered. Alcohol significantly contributed to the equation and increased the variance accounted for to 18.9%.

In the fifth step, both mood variables were entered. Taken together, depression and anger were not significant. The overall model remained significant, with 25.4% of the variance was accounted for.

In the sixth and final step, problem-solving was entered. This variable was not significant. The overall model remained significant, and the total variance accounted for increased only slightly, to 25.7%.

Table 20. Variables entered in the stepwise logistic regression analysis for smoking status with outlier removed

Variable	Step χ^2	df	Sig.	Model χ^2	Df	Sig.	Nakelkerke R^2
Demographics	6.528	2	.0382				.089
Diagnosis	.019	1	.8902	6.547	3	.0878	.089
Smoking history	1.442	2	.4862	7.990	5	.1568	.108
Alcohol	6.442	1	.0111	14.431	6	.0252	.189
Mood	5.410	2	.0669	19.841	8	.0110	.254
Problem solving	.314	1	.5752	20.155	9	.0170	.257

* $p < .05$

Next, independent variables that were specifically predictive of smoking status were examined. A list of variables included in the final equation is presented in Table 21. In the final equation, marital status, alcohol use, and depression were significant.

Table 21. Variables in the equation

Variable	B	S.E.	Wald	Df	Sig.	R	Exp(B)	95.0% C.I. for Exp(B)	
								Lower	Upper
Marital 4 [a]			9.0010	2	.0111*	.2215			
Marital4 (1) [b]	-.1161	1.0190	.0130	1	.9093	.0000	.8904	.1208	6.5614
Marital4 (2) [c]	1.7045	.5869	8.4347	1	.0037	.2512	5.4985	1.7405	17.3699
Diagnosis	-.4229	.5325	.6305	1	.4272	.0000	.6552	.2307	1.8606
Pack years	.0080	.0089	.8060	1	.3693	.0000	1.0080	.9906	1.0258
Nicotine dependence [d]	-.1249	.5867	.0453	1	.8314	.0000	.8826	.2795	2.7873
ETOH	.0919	.0431	4.5481	1	.0330*	.1581	1.0963	1.0075	1.1929
POMS Depr	-.0831	.0419	3.9274	1	.0475*	-.1375	.9203	.8477	.9991
POMS Ang	.0996	.0500	3.9645	1	.5774	.1388	1.1047	1.0016	1.2185
SPSI Total	.0544	.0976	.3104	1	.2502	.0000	1.0559	.8720	1.2786
Constant	-2.2929	1.9940	1.3224	1					

[a] married or living with significant other. [b] single. [c] separated, divorced, or widowed. [d] measured by the question "how soon after you wake/woke up do/did you smoke your first cigarette?"

* $p < .05$

While this model is only moderately predictive of smoking status (R^2 = 25.7%), it is still useful as a method to classify patients into smoking status groups. The classification table (see Table 22) for the final model shows an overall correct classification of 80% of the cases. This is only slightly higher than the base-rate classification of 72% (i.e., if nothing was known about a patient, he or she would be classified into the larger category of former smokers). Moreover, 13 current smokers were correctly classified using the model, and 16 current smokers were mis-classified as former smokers, producing a sensitivity rate of only 44.83% to detect current smokers.

Table 22. Classification table for final model

Observed	Predicted		Percentage Correct
	Former Smoker	Current Smoker	
Former Smoker	69	4	94.52
Current Smoker	16	13	44.83
Overall Percentage			80.39

Note. The cut value is .500

Analysis of Response Operating Characteristics

Response operating characteristics (ROC) analysis is often used to improve classification. In the above model, the final classification rate is 80.4%, with slightly less than chance classification of current smokers (44.8%). As Table 22 reveals, 16 out of 29 (55.2%) current smokers are misclassified as former smokers. This model assumes equal odds at baseline for being a current versus former smoker. However, since we know that at baseline the odds are more likely to be a former smoker, the ROC analysis allows us to change the cut value accordingly and improve the sensitivity (correctly identifying current smokers; 44.8%) and specificity (correctly ruling out current smokers, or identifying former smokers; 94.5%). To change the cut point, a ROC analysis is conducted.

The first step in the ROC analysis is to examine the ROC curve (see Figure 1) and its coordinates, looking for a line where sensitivity and specificity are both high. The straight line represents a model in which the variables have no relationship to the outcome. Each point on the curved line represents a possible cut point. Horizontal lines along the curve represent cut points in which we can keep the sensitivity the same but improve specificity, and vertical lines represent cut points where we can keep specificity the same and improve sensitivity. One way to describe the curve is to present the area under the curve. An area under the curve of .5 would means that there is no ability to predict outcome whereas an area of 1.0 represents a model with perfect prediction. Thus, the farther away the curve is from the straight line, the stronger the model. The area under the curve for this model is 74.4.

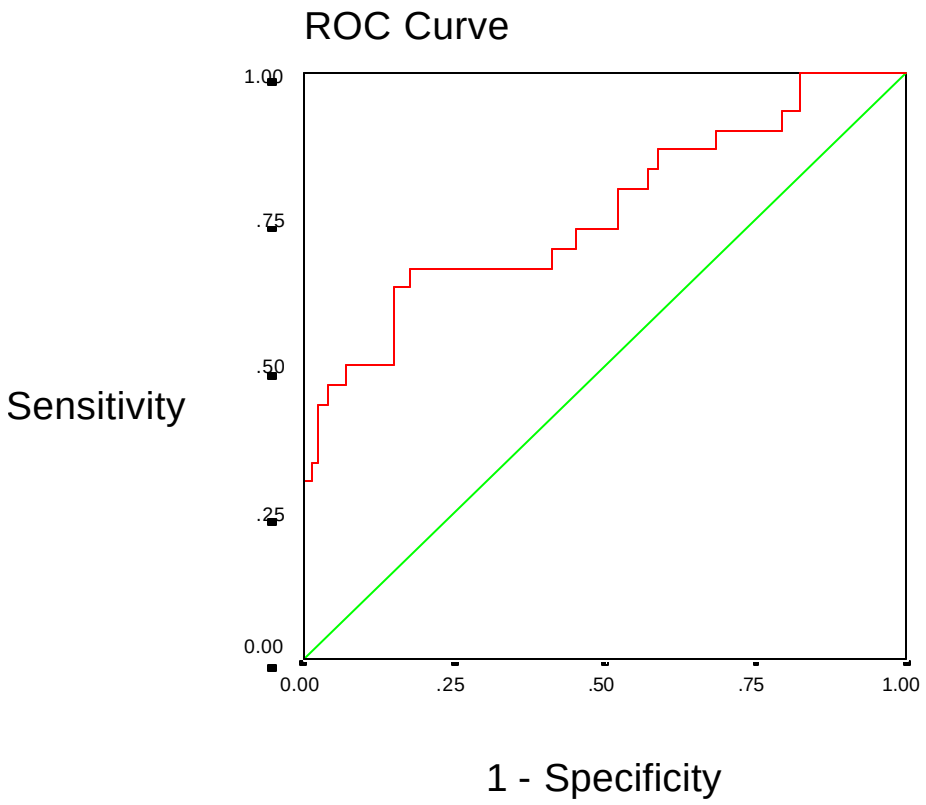

Figure 1. Curve for response operating characteristics

The second step involves determining a cut-point by examining the table of possible cut-points that are represented by the curve. A list of some of the possible cut-points, along with their corresponding sensitivity and specificity, is presented in Table 23. A ROC analysis which increases the sensitivity of the model would yield a more clinically meaningful and useful model. A cut point which increases the sensitivity of the model without sacrificing too much specificity would be a cut point equal to .2803. This cut point yields a sensitivity of 69% and a specificity of 71.2%. A ROC analysis using a cut point of .2803 rather than .500 yields a new classification table (see Table 24). While the overall classification of the model reduces from 80% to 70%, sensitivity has significantly increased. In other words, the model allows us to correctly identify more current smokers and misclassify fewer current smokers.

Table 23. Coordinates of the ROC curve

Cut point	Sensitivity	1 – Specificity
.0457	1.000	.973
.0908	.966	.877
.1359	.793	.671
.1928	.793	.425
.2146	.690	.425
.2642	.690	.301
.2803	.690	.288
.5026	.448	.055
.5446	.414	.000
.8757	.034	.000

Table 24. Classification table for final model based on ROC analysis

Observed	Predicted		Percentage Correct
	Former Smoker	Current Smoker	
Former Smoker	52	21	71.23
Current Smoker	9	20	68.97
Overall Percentage			70.59

Note. The cut value is .2803

Secondary Analysis

As discussed earlier, one of the initial research questions included examining the role of medical severity in predicting smoking status within the COPD population as well as in the head and neck cancer population. In order to increase the power of the statistical analysis, the two medical populations were combined to increase the sample size. However, because severity ratings for these two populations differ, this predictor variable was eliminated from the statistical analysis. Therefore, a secondary, post-hoc-like analysis was conducted in order to examine the relationship between disease severity and smoking status within each group. In the head and neck cancer group, smoking status was not related to stage of cancer at initial diagnosis ($\chi 2(4) = 4.718$; $p = .317$). Frequencies of current smokers and former smokers according to TNM stage in the head and neck cancer group is presented in Table 25. In the COPD group, there was a significant difference between the current smokers and the former smokers across diagnostic groups ($\chi 2(2) = 9.579$; $p = .008$). Frequencies of current smokers and former smokers according to medical severity in the COPD group are presented in Table 26.

Table 25. Frequencies of current smokers and former smokers in the head and neck cancer group according to TNM stage at initial diagnosis

TNM Stage	Current smokers	Former smokers
I	4	7
II	4	6
III	1	11
IV	4	9

Table 26. Frequencies of current smokers and former smokers in the COPD group according to FEV1%

FEV1% predicted	Current smokers	Former smokers
50-70	10	8
35-49	6	14
<35	1	15

CHAPTER 14: POWER ANALYSIS

Statistical power is defined as "the probability of correctly rejecting the null hypothesis when it should be rejected" (Hair et al., 1995, p. 10). Statistical power is a function of three variables: effect size, alpha, and sample size. Effect size is a theoretical construct, and is an "estimate of the degree to which the phenomenon being studied (e.g., correlation or difference in means) exists in the population" (Hair et al., 1995, p. 1). Alpha, also known as Type I error, is the "probability of rejecting the null hypothesis when the null hypothesis is actually true" (Hair et al., 1995, p. 10). In other words, alpha is a false positive. "Conventional guidelines suggest alpha levels of .05 or .01" (Hair et al., 1995, p. 11). As alpha decreases (i.e., becomes more conservative), power also decreases. Sample size is integral to statistical power because as the sample size decreases, the power to detect significant differences also decreases. A sample that is too small may miss significant findings, whereas when a sample is extremely large, the analysis can be oversensitive, finding any differences between groups to be significant.

In this study, a total of 103 participants were accrued whose data was analyzed. As discussed in the Rationale section, to date, there is minimal research investigating the psychosocial variables related to smoking behavior in these two medical populations. Therefore, this study, and the logistic regression analysis, are exploratory in nature. In order to build the logistic regression model, relationships between each independent variable and the outcome variable were investigated through bivariate correlations and t-tests. In order to

determine the power of the statistical analysis, a medium effect size was selected, since there is a dearth of literature from which to suggest otherwise. As is convention, alpha was set at .05. Assuming a medium effect size and an alpha equal to .05, this study needed 50 subjects per group to yield a 70% power for t-tests, and 30 subjects per group to yield a 90% power for bivariate correlations (Cohen, 1977). Therefore, there was sufficient power to detect relationships between each independent variable and the outcome variable, smoking status.

There is not enough information from prior studies to run a meaningful power analysis for a logistic regression for an exploratory study such as this one. By convention, a total of at least 100 participants is recommended for studies where multivariate techniques are used, as is approximately 20-40 subjects per independent variable in a regression analysis. Thus, given that nine independent variables were included in the final model, the lack of statistically significant findings may be due to a lack of power to detect differences. This lack of power is a result of the relatively small sample size for the multivariate model, and may also be related to a smaller effect size than estimated in the power analysis.

CHAPTER 15: DISCUSSION

The strength of this study lies in its examination of psychosocial predictors of smoking behavior in people diagnosed with either head and neck cancer or COPD. To date, there is very little research investigating the relationship between mood (specifically, depression and anger) and smoking behavior and problem-solving skills and smoking behavior, in these two medical populations. The goal of the present study was to examine whether depression, anger and problem-solving skills predict smoking status in a group of patients diagnosed with either head and neck cancer or COPD.

In this study, 68.5% (37/54) of individuals with COPD and 73.5% (36/49) of individuals with head and neck cancer were classified as former smokers, and the difference in the quit rates between the two medical populations was nonsignificant. Contrary to expectation, diagnosis did not predict smoking behavior in these two medical populations. Epidemiologic research has reported that approximately 30% of individuals with COPD (Anthonisen et al., 1994; Green, 1995) and approximately 70% of individuals with head and neck cancer (Ostroff et al., 1995; Spitz et al., 1990) quit smoking upon a diagnosis. Therefore, while the cessation rates for the head and neck cancer group are consistent with previous findings, the cessation rate of the COPD group is substantially higher.

There are several reasons why this study may have yielded a higher cessation rate in the COPD population than other studies of the COPD population. First, many of the COPD patients who participated in this study attend the Temple University Hospital Lung Center.

This center conducts a variety of treatment outcome studies with people with COPD, including lung volume reduction surgery and lung transplantation. Individuals with severe COPD are required to discontinue smoking tobacco in order to qualify for these treatments. Therefore, not only may these individuals have more motivation to quit, but they may also receive more advice and assistance from their pulmonary physician. Second, although confidentiality of the patient's smoking status was emphasized, individuals who continue to smoke may still feel compelled to under-report their current smoking habits or misreport their current smoking status. Because biologic assays were not taken, there is no means by which to confirm the validity of the participants' responses. Therefore, there may be a high rate of false negatives (e.g., current smokers who claim to have quit). Third, the sample size of this study is relatively small compared to large population studies, and therefore, may be too small to reflect epidemiological cessation rates. Fourth, a point prevalence measure of smoking status may not accurately reflect smoking status because of the limitations of this type of measurement. More specifically, smoking cigarettes is an ever-changing behavior. Often, individuals attempt to quit many times before being successful abstainers over the long-term. In fact, "most people who are attempting to quit smoking change their status more often than once a year" (Murray et al., 1998, p. 1324). Therefore, it is possible that patients have accurately reported their smoking status if they have temporarily stopped smoking cigarettes. Redefining the classification for diagnosis could potentially alter the results of the study. For instance, in this study, current smokers included only individuals who smoked cigarettes within the past 7 days. Redefining current smokers to include anyone who smoked

within the past month could yield different findings. This is unlikely, however, given the follow-up results. A follow-up interview over the telephone found that of the individuals reached for follow-up, 100% of participants remained in the same smoking status category as when they were recruited into the study. Finally, the higher cessation rates of this COPD population as compared to previous literature may be a reflection of selection bias. Current smokers may be more likely to decline participation in the study for fear that their physician will learn of their smoking behavior.

Research investigating the relationship between demographic variables and smoking behavior in the general, COPD, and cancer populations has been mixed. The typical demographic variables that have been previously investigated include age, gender, marital status, and income. In this study, income was eliminated from the analysis due to insufficient data (i.e., many participants refused to report their income). Overall, in this sample, demographic variables, taken together, did not predict whether an individual continues to smoke or quits smoking upon a diagnosis. The only demographic variable found to be significantly predictive of smoking status was marital status. Because research on demographic variables has resulted in varied results in the past, the results of this study neither confirm nor disconfirm previous findings. Given the age of the population ranged from 41-80, with a mean age of 60, it would be highly unlikely for age to predict smoking status, as previous studies demonstrating an age difference have included samples with a much larger age range. The fact that marital status was found predictive of smoking status confirms previous studies investigating the role of social support. However, many of these studies have

focused on the relationship between smoking behavior and number of other smokers in the household (Matheny & Weatherman, 1998; McIntyre-Kingsolver, 1986). However, in this study, marital status and number of other smokers in the household was not correlated. Furthermore, the median number of other smokers in the household was zero.

This study found that neither pack years nor nicotine dependence improved prediction of smoking status. This finding was unexpected, as several studies have found that amount smoked (Gritz, 1993; Salive, 1992; Spitz, 1990) and nicotine dependence (Cohen, 1989; Duncan, 1992) are associated with smoking behavior. There are several reasons why this study may have resulted in different findings. First, although some authors suggest that "time to first cigarette" is a valid measure of nicotine dependence (Gritz, 1993; Hall et al., 1985; Velicer, 1992), this may not be the case. Nicotine dependence may be a more complex construct. Second, asking former smokers how soon they smoked their first cigarette upon waking in the morning relies on the participant's memory. Retrospective data can be intentionally, or unintentionally, false. A third alternative to the lack of findings is that there are a variety of other smoking history variables which may be predictive, but were not examined in this study. Several of the smoking history variables which were not included in the logistic regression analysis include number of previous quit attempts, duration of previous quit attempts, and number of other smokers in the household. Although these questions were asked of the participants, the questions were not specific enough, and therefore, did not accurately reflect the construct the researcher was attempting to measure. For instance, when reviewing the data, it was unclear whether the number of previous quit attempts

included the current quit attempt. Likewise, current number of other smokers in the household was assessed rather than number of additional smokers in the household at the time the participant (if former smoker) had quit smoking. Therefore, it is possible that the smoking history variables which were not examined may in fact be significant predictors.

In this study, alcohol use was predictive of smoking status. While this is consistent with the literature, the relevance of this variable is questionable. More specifically, current alcohol use was assessed rather than alcohol use at the time of diagnosis or at the time of cessation (for former smokers). This reduces the relevance of the interpretation of this variable for several reasons. First, individuals who quit tobacco often quit alcohol use at the same time. Therefore, it makes sense that former smokers will report decreased alcohol use as compared to current smokers. Moreover, studies show that for individuals who have both habits, individuals are more likely to give up alcohol before smoking (Deleyiannis et al., 1996).

Depression improved prediction of smoking status in this study. This finding is consistent with previous research of smoking behavior in the general population. An overwhelming amount of research suggests that depression is highly correlated with smoking behavior (Coogan, 1998; Glassman et al., 1990; Wang, 1994), that depression is associated with smoking cessation (Anda et al., 1990; Ginsberg, 1995; Glassman, 1993; Hall et al., 1993; Rabois & Haaga, 1997), and that depression predicts relapse (Doherty et al., 1995; Shiffman, 1986; Shiffman et al., 1997; Swan, 1996). A strength of the present study is that it specifically examines the relationship between mood and smoking behavior in two medical

groups in which smoking behavior is directly linked to diagnosis, treatment outcome, morbidity, and mortality. There are few previous studies investigating the relationship between depression and smoking behavior in the head and neck cancer or COPD population. Therefore, the finding in this study that depression predicts smoking status in this combined group provides interesting preliminary results. Despite the relevance of this information, there are several limitations to the interpretation of this finding. First, depression was not assessed at the time of diagnosis, nor for former smokers, at the time of cessation. In other words, "fluctuations in mood more proximal to high-risk situations were not assessed" (Hall et al., 1994, p. 145). Depressive symptoms can change over time. Therefore, whether a participant is or is not depressed at the time of participation in this study does not indicate whether the individual was depressed, or would have reported symptoms of depression, at the time of diagnosis or at the time of cessation. For instance, depression could theoretically increase at time of diagnosis, and then increase or decrease over time depending on quality of life changes, treatment outcome, and a variety of other variables that were not assessed.

Anger was not found to predict smoking status in this study. Initial studies have found an association between anger and smoking behavior in the general population (Doherty et al., 1995; Ginsberg et al., 1995; Speilberg et al., 1998; Tate et al., 1996; Tsuda et al., 1996). Similar to depression, anger is a dynamic construct. Anger was assessed at the time of the participant's participation in the study, rather than at the time the participant (if former smoker) quit smoking cigarettes. Current symptoms of anger do not necessarily reflect the

participant's experience of anger at the time of cessation. This is particularly relevant because in the short-term, abstinence leads to increased symptoms of anger (Doherty et al., 1995; Ginsberg et al., 1995; Tate et al., 1996; Tsuda et al., 1996).

Finally, problem-solving skills were not found to predict smoking status. This finding is contrary to previous research suggesting that former smokers report better coping skills than current smokers (Horowitz, 1985; Matheny & Weatherman, 1998; Wewers, 1988). There are several reasons why this study yielded different findings. First, the problem-solving construct (SPSI-R) used in this study may be testing different constructs than previous studies. This is the first time problem solving, as defined by the SPSI-R, has been examined with smoking behavior.

Second, problem-solving skills were assessed at the time of participation, not at the time the individual (i.e., former smoker) quit smoking. Although it is unlikely that an individual's problem-solving skills would change over time without an intervention (i.e., problem-solving skills training), the influence of an individual's successful quit attempt on their problem-solving skills (in particular, problem orientation), cannot be ruled out.

Third, the effect size for problem solving may be smaller than initially perceived. If so, a much larger sample may be required in order to detect a significant relationship between problem-solving and smoking behavior.

Fourth, perhaps it is not an individual's overall problem-solving ability which is associated with smoking status, but rather, one of the component problem-solving skills. For instance, previous research on coping skills in the smoking literature suggests that individuals

who demonstrated less avoidance behavior were more successful abstainers (Horowitz, 1985). Therefore, perhaps one of the components of problem solving (e.g., subscales of the SPSI) is predictive of smoking status. Specifically, problem solving can help to increase one's sense of self-efficacy and also help individuals to better define their problems (e.g. recognize cues; determine which situations are triggers), and brainstorm different ways to not only reach one's goals, but to also overcome obstacles to prevent relapse. However, exploratory follow-up analyses of the SPSI subscales demonstrated low correlations between the subscales and smoking status. Therefore, it is unlikely that these variables would be significant predictors in the logistic regression analysis.

Fifth, problem-solving skills may not be a predictor or smoking status, but rather, may moderate the relationship between mood (e.g., depression, anger) and smoking status. Much of the previous literature demonstrates the moderating role of problem solving on stress-related depression (Nezu, 1985, 1986, 1987; Nezu et al., 1986; Nezu & Ronan, 1985).

Finally, it may be that problem-solving ability is not correlated with smoking behavior in this sample, or in these medical populations. Several other theories of health behavior (e.g., Health Belief Model, Transtheoretical Model of Change, Theory of Self-Efficacy) were highlighted earlier. Although these theories were not specifically tested in this study, it is possible that these variables play a stronger role in smoking behavior and smoking cessation, and therefore, cannot be disregarded.

CHAPTER 16: LIMITATIONS

In addition to the limitations highlighted in the Discussion section, there are several additional limitations to this study, related to internal and external validity, measurement problems, and problems with statistical analysis.

First, this study is a cross sectional design, prohibiting a causal interpretation of the data. We cannot infer that depression predicts future smoking behavior, as depression and smoking status were assessed at the same point in time. Furthermore, this study cannot rule out the possibility that smoking behavior (i.e., smoking cessation) does not have a causal effect on the predictor variables. For instance, individuals who successfully quit smoking may subsequently fare better from their treatment, and thus, experience a decrease in depression. Likewise, smoking may affect an individual's orientation such that "failing to quit smoking strengthens a person's belief that s/he cannot control her/his life" (Stronks et al., 1997).

Second, this study lacks external validity because the generalizeability of the results is limited. The information gleaned about smoking behaviors from this study is limited to head and neck cancer and COPD. Predictors of smoking behavior cannot be generalized to other cancers or respiratory diseases. This is not much of a concern, as the interest of the research was specifically smoking behavior in these two particular medical populations.

Third, for the former smokers, smoking behavior questions relied on retrospective memory. Retrospective data is more likely to be inaccurate. The reliability of their answers depends on the accuracy of their memory. Furthermore, the greater the time between the

former smoker's quit date and the time of their participation in the study, the greater the likelihood of mis-reporting. Therefore, smoking variables which are used in the statistical analyses, such as pack years, or the response to the question "how soon after you woke up would you smoke your first cigarette" may be inaccurate, and subsequently impact the results. Even if participants attempted to provide honest reports, there is no means by which to determine whether the responses of former smokers are inaccurate, and if inaccurate, the extent to which these participants underreported or over-reported their smoking behaviors and alcohol use.

Fourth, smoking behavior is a dynamic process. In this study, smoking status is a point prevalence measure. Therefore, the smoking rates may be inaccurate, and may underestimate the number of smokers. For instance, individuals who were classified as former smokers may have just quit in the past week and may relapse next week. However, time to relapse is quite variable, so that determining a time for follow-up assessment is difficult. Some researchers suggest that the most difficult time for smokers, when there are the strongest urges to smoke, is immediately after quitting. However, other researchers claim that a typical smoker starts up 3 months after quitting. In this study, smoking status was assessed twice: at initial entrance into the study, and a second time 3 months later. Individuals who remained abstinent were classified as former smokers.

Fifth, measurement of variables which posed a difficulty particular to the head and neck cancer group were those that inquired about withdrawal symptoms, such as craving, irritability, anxiety, increased eating and restlessness. Individuals with head and neck cancer

who do quit, often quit the day of their surgery. Therefore, they may experience the afore-mentioned withdrawal symptoms, although not necessarily due to withdrawal from nicotine. Furthermore, these individuals are unlikely to report increased eating, as they are unable to eat solid food for many months post surgery.

Sixth, measurement of alcohol use in this study could be improved. Current alcohol use, but not alcohol history, was assessed. Many individuals quit alcohol use along with smoking cessation. Therefore, current alcohol use is not reflective of prior alcohol use. A more time-specific assessment might alter the results of the analysis.

Seventh, although classification of smoking status was chosen based on previous research, classification remains arbitrary. Many studies use a cut-off of 7 days, such that individuals who have smoked within the past 7 days are considered current smokers. A different cut-off point might yield different results. Also, this researcher considered smoking even a puff of a cigarette as smoking behavior. Some might argue that there is a difference between an individual who smoked one puff in the past 7 days and an individual who smoked one pack of cigarettes within the past 7 days. For instance, one might argue that the former is not nicotine dependent and the latter is, and that the psychosocial profile might differ since depression and nicotine dependence share some of the same biologic underpinnings. Therefore, a different classification system could yield different results.

In addition to measurement limitations and difficulties, there are several limitations related to the patient population. First, the pool of participants may be a reflection of selection bias. Current smokers may be more likely to decline participating in the study for

fear of their physician learning of their continued habit, or due to embarrassment at continuing to do a behavior that is considered socially unacceptable. Second, even participants who do complete the study may underreport their smoking history or current smoking status. In this study, underreporting of current smoking habits might not have affected the data since participants are being classified as either current smokers or former smokers. Underreporting only becomes a problem for the analysis if the participant actually claims to be a former smoker when he or she is, in fact, a current smoker. Although a biochemical assay would be desirable, self-report measures are the primary measures used for assessing smoking status in most investigations (Matheny and Weatherman, 1998; Ossip-Klein et al., 1986). Furthermore, the debate as to whether individuals underreport their smoking or whether their self-reports are accurate has not been resolved. Even if participants attempt to provide honest reports, there may be error, as highlighted above, due simply to retrospective bias. Persson and Norell (1989) found that those who had decreased their consumption over time underestimated previous cigarette consumption, whereas those who did not change their consumption provided more accurate retrospective information.

Data on depression, anger and problem-solving skills were also solely based on self-reports. It may be helpful to confirm self-reported symptoms and increase the reliability of the findings by utilizing clinician ratings or reports by significant others.

Finally, due to the small sample size and the change in research analysis, this study was unable to answer one of the initial research questions investigating the role of medical severity in predicting smoking status of two separate medical populations. Thus, one of the

hypothesized predictors was eliminated from the analysis. The analysis of the combined groups does not allow for interpretation of either group alone. Although the power was too low to conduct a statistical analysis of the medical groups separately, medical severity may be a significant predictor of smoking status, and therefore, cannot be ruled out as a potential predictor.

CHAPTER 17: FUTURE RESEARCH

Based on the results, and on the limitations, of this study, several recommendations can be made for future research.

Given the previous research that medical severity impacts smoking behavior, and given the results of this study suggesting that measures of pulmonary function are associated with smoking status, more information could be gleaned from two separate studies, one on smoking behavior in the COPD population, and one on smoking behavior in the head and neck cancer population. A much larger sample size would be required in order to detect differences in smoking behavior.

Research incorporating a longitudinal design is essential. This study incorporated a cross-sectional design to answer temporal questions. Therefore, the results can only suggest that current depression is associated with current smoking status. The results cannot make any inferences about a causal relationship. Therefore, a study in which individuals are assessed at the time of their diagnosis, and then followed over time, will better answer the research questions of this study.

Moreover, because smoking behavior is a dynamic process, and because individuals will attempt to quit smoking several times before they are successful over the long-term, a study which assesses smoking status over the course of years, rather than months, may better capture the dynamic nature of smoking behavior, and may also more correctly classify smokers.

Several of the important predictor variables, as well as the main outcome

variable, in this study, were based solely on self-reports. Multiple assessments of mood, problem-solving skills, and smoking status, from different sources, would be more reliable. For instance, future research could incorporate a self-report measure of mood as well as a clinician rating, and smoking status could be confirmed by a biochemical validation measure.

The relationship between problem solving and smoking behavior warrants further investigation. The fact that problem-solving skills did not predict smoking status in this study does not rule out the possibility that problem-solving skills and smoking behavior are related in another way. Given the strong findings from previous research of the moderating role of problem solving on stress-related depression, a promising area of research is the examination of problem solving as a moderator of depression and smoking behavior.

Two additional psychosocial variables that would be important to investigate in future studies are anxiety and hostility. Some studies have found an association between anxiety and smoking behavior (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Gilbert, McClernon, Rabinovich, Plath, Masson, Anderson, et al., 2002), whereas other studies have not found anxiety-related differences (Takemura, Akanuma, Kikuchi, & Inaba, 1999; Zvolensky, Feldner, Eifert, & Brown, 2001). For instance, in a group of 60 smokers with a past history of Major Depressive Disorder, higher anxiety (as measured by the Anxiety Sensitivity Index) during smoking cessation was associated with an increased risk of relapse during the first seven days (Brown et al., 2001). Likewise, in an intervention study of 96 female smokers, anxiety (as measured by the Beck Anxiety Inventory) increased during the initial quit attempt, and then returned to baseline levels after one week (Gilbert et al., 2002).

Given that anxiety is often reported as a symptom of withdrawal, and given some of the previous research, this variable might yield improtnat information regarding smoking behavior.

In addition to anxiety, smoking behavior has been associated with hostility (Calhoun et al., 2001; Janner et al., 1999; Scherwitz & Rugulis, 1991; Whiteman et al., 1997; Williams & Williams). For instance, Janner et al. (1999) found nicotine reduced reports of anger (as reported in an ambulatory diary) in both smokers and nonsmokers with high hostility (as measured by the Cook-Medley Hostility subscale of the Multiphasic Personality Inventory [MMPI]), but not in those with low hostility. Additionally, they found that in a group of smokers and nonsmokers with high hostility on the nicotine patch, nicotine reduced reports of anger in both smokers and nonsmokers, suggesting that reports of anger were not simply a symptom of withdrawal (Janner et al., 1999). The current study assessed this variable using the anger/hostility subscale of the POMS. The POMS assesses an individual's mood over the past week, and therefore, reflects more of a state anger. In addition, some researchers suggest that there is a difference between anger and hostility such that hostility is a trait characterized by a cynical mistrust of others, a low threshold for anger, and the aggressive expression of anger (Barefoot, Peterson, Dahlstrom, Siegler, Anderson, & Williams, 1991), whereas anger is the mood component of the hostility trait. Therefore, a measure which better captures the hostility trait might yield important information. For instance, previous studies have used the Cook-Medley Hostility subscale of the MMPI (

Calhoun et al., 2001; Scherwitz & Rugulies, 1991) and the Bedford-Foulds Personality Deviance Questionnaire (Whiteman et al., 1997) to measure hostility.

Finally, a study which includes all patients, including those who never smoked, might yield valuable information. The current study accepted only those who were smoking cigarettes at the time of their diagnosis. Based on this criteria, 61 people were excluded from the study because they had never smoked cigarettes, and 83 people were excluded from the study because they had quit prior to their diagnosis. Future research could include these populations as well, as they might prove to be interesting comparisons groups. Understanding these two additional populations might further explain smoking behaviors in these medical populations.

The results of this study have important implications for the role of clinical health psychology in smoking cessation. Although the smoking cessation rates for both the COPD population and the head and neck cancer population in this study were quite high (approximately 70%), a significant proportion of patients continue to smoke cigarettes despite their diagnosis. Helping these patients quit smoking has important implications for treatment outcome. More specifically, individuals who quit smoking respond to treatment better, experience slower disease progression, and have decreased mortality rates. Therefore it is important to identify patients at risk for continued smoking and target those individuals with treatment aimed at reducing barriers to smoking cessation.

This study demonstrates an association between depression and smoking status such that those who report symptoms of depression are more likely to continue smoking after a

diagnosis of either head and neck cancer or COPD. It may be useful, therefore, to identify patients who are depressed, and to provide them with psychotherapeutic intervention in order to reduce their depressive symptoms and to subsequently improve the chance of a successful cessation.

CHAPTER 18: SUMMARY

The present study examined psychosocial predictors of smoking status in a group of individuals diagnosed with either head and neck cancer or COPD. Results demonstrated that marital status, alcohol use, and depression predict smoking status above and beyond demographic variables. Results also demonstrated that smoking history, anger, and problem-solving skills did not predict smoking status in this population.

Sixty-eight percent of individuals with COPD and 75% of individuals with head and neck cancer quit smoking since their initial diagnosis. Medical severity appeared to be associated with smoking status in the COPD population, but not in those diagnosed with head and neck cancer.

The fact that depression increased prediction of smoking status suggests that interventions targeted to treat depression may help individuals successfully quit smoking. Such treatments may, subsequently, reduce morbidity and mortality due to head and neck cancer and to COPD.

LIST OF REFERENCES

Abrams, D.B., Follick, M.J., Biener, L., Carey, K.B., & Hitti, J. (1987). Saliva cotinine as a measure of smoking status in field settings. *American Journal of Public Health, 77*(7), 846-848.

Agle, D.P., Baum, G.L., Chester, E.H., & Wendt, M. (1973). Multidisciplinary treatment of chronic pulmonary insufficiency: 1. Psychologic aspects of rehabilitation. P*sychosomatic Medicine, 35*(1), 41 – 49.

American Cancer Society. (1994). *Cancer facts & figures - 1994*. Atlanta: Author.

American Thoracic Society. (1995). Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *American Journal of Respiratory Critical Care Medicine. 152*(5), S77-S120.

Anda, R.F., Williamson, D.F., Escobedo, L.G., Mast, E.E., Giovino, G.A., & Remington, P.L. (1990). Depression and the dynamics of smoking: A national perspective. *Journal of the American Medical Association, 264*(12), 1541-1545.

Anthonisen, N.R., Connett, J.E., Kiley, J.P., Altose, M.D., Bailey, W.C., Buist, A.S. et al. (1994). Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1: The Lung Health Study. *JAMA, 272*(19), 1497-1505.

Baer, J.S., Holt, C.S.., & Lichtenstein, E. (1986). Self-efficacy and smoking reexamined: Construct validity and clinical utility. *Journal of Consulting and Clinical Psychology, 54*(6), 846-852.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84(2), 191-215.

Barefoot, J.C., Peterson, B.L., Dahlstrom, W.G., Siegler, I.C., Anderson, N.B., and Williams, Jr., R.B. (1991). Hostility patterns and health implications: Correlates of Cook-Medley Hostility scale scores in a national survey. *Health Psychology, 10*, 18-24.

Becker, M.H., Haefner, D.P., Kasl, S.V., Kirscht, J.P., Maiman, L.A., & Rosenstock,

I.M. (1977). Selected psychosocial models and correlates of individuals health-related behaviors. *Medical Care, 15*(5), 27-46.

Bennett, P, Norman, P., Moore, L. & Murphy, S. (1997). Health locus of control and value for health in smokers and nonsmokers. *Health Psychology, 16*(2), 179-182.

Bjornson, W., Rand, C., Connett, J.E., Lindgern, P., Nides, M., Popoe, F. et al. (1995). Gender differences in smoking cessation after 3 years in the Lung Health Study. *American Journal of Public Health, 85*, 223-230.

Blot, W.J., McLaughlin, J.K., Winn, D.M., Austin, D.F., Greenberg, R.S., Preston-Martin, S., et al. (1988). Smoking and drinking in relation to oral and pharyngeal cancer. *Cancer Research, 48*, 3282-3287.

Bosetti, C., Franceschi, S., Levi, F., Negri, E., Talamini, R., & La Vecchia, C. (2000). Smoking and drinking cessation and the risk of aesophageal cancer. *British Journal of Cancer, 83*(5), 689-691.

Brennan, JA., Boyle, JO, Koch, WM, Goodman, SN., Hruban, RH, Eby, YJ, et al., (1995). Association between cigarette smoking and mutation of the p53 gene in squamous-cell carcinoma of the head and neck. *The New England Journal of Medicine, 332*(11), 712-717.

Breslau, N., Kilbey, M., & Andreski, P. (1993). Vulnerability to psychopathology in nicotine-dependent smokers: An epidemiologic study of young adults. *American Journal of Psychiatry, 150*(6), 941-946.

Browman, G.P., Wong, G., Hodson, I., Sathya, J., Russell, R., McAlpine, L., Skingley, P., et al. (1993). Influence of cigarette smoking on the efficacy of radiation therapy in head and neck cancer. *The New England Journal of Medicine, 328*(3), 159-163.

Brownson, R.C., Jackson-Thomson, J., Wilkerson, J.C., Davis, J.R., Ownes, N.W., & Fisher, E.B. (1992). Demographic and socioeconomic differences in beliefs about the health effects of smoking. *American Journal of Public Health, 82*(1), 99-103.

Bunch J.M. & Schneider H.G. (1991). Smoking-specific locus of control. P*sychological Reports, 69*, 1075-1081.

Bundgaard, T., Wildt, J., Frydenberg, M., Elbrond, O., & Nielsen, J.E. (1995). Case-control study of squamous cell cancer of the oral cavity in Denmark. *Cancer Causes and Control, 6*, 57-67.

Carbone, D. (1992). Smoking and cancer. *The American Journal of Medicine, 93* (Suppl. 1A), 13S-17S.

Castellsague, X. (1999). Cancer of the oral cavity and pharynx in nonsmokers who drink alcohol and in nondrinkers who smoke tobacco. *Journal of the National Cancer Institute, 91*(15), 1336-1337.

Castellsague, X., Munoz, N. De Stefani, E., Victoria, C.G., Castelletto, R., Rolon, P.A. & Quintana, J. (1999). Independent and joint effects of tobacco smoking and alcohol drinking on the risk of esophageal cancer in men and women. *International Journal of Cancer, 82*, 657-664.

Castellsague, X., Munoz, N., De Stefani, E., Victora, C.G., Quintana, M. J., Castelletto, R., Rolon, P.A., et al. (2000). Smoking and drinking cessation and risk of esophageal cancer (Spain). *Cancer Causes and Control, 11*, 813-818.

Centers for Disease Control (1991). Smoking-attributable mortality and years of potential life lost - United States, 1988. Centers for Disease Control; 1991. *Morbidity and Mortality Weekly Report, 40*, 62-63, 69-71.

Centers for Disease Control and Prevention (1993). Smoking-attributable mortality and years of potential life lost - United States, 1990. *Morbidity and Mortality Weekly Report, 42*(33): 645-648.

Centers for Disease Control (1997). Cigarettes smoking among adults-United States, 1995. *Morbidity and Mortality Weekly Report, 46*(5), 1217-1220.

Centers for Disease Control and Prevention. (1999). Cigarette smoking among adults-United States, 1997. *Morbidity and Mortality Weekly Report, 48*(43), 994-996.

Cinciripini, P.M., Wetter, D.W., & McClure, J.B. (1997). Scheduled reduced smoking: Effects on smoking abstinence and potential mechanisms of action. *Addictive Behaviors, 22*(6), 759-767.

Cohen, J. (1977). *Statistical power analysis for the behavioral* sciences (Rev. ed.). New York: Academic Press.

Cohen, S., Lichtenstein, E., Prochaska, J.O., Rossi, J.S., Gritz, E.R., Carr, C., et al. (1989). Debunking myths about self-quitting: evidence from 10 prospective studies of person who attempt to quit smoking by themselves. *American Psychologist, 44*(11), 1355-1365.

Colletti, G. & Kopel, S.A. (1979). Maintaining behavior change: An investigation of three maintenance strategies and the relationship of self-attribution to long-term reduction of cigarette smoking. Journal *of Consulting and Clinical Psychology, 47*(3), 614-617.

Colletti, G, Supnick, JA., & Payne, TJ. (1985). The smoking self-efficacy questionnaire (SSEQ): Preliminary scale development and validation. *Behavioral Assessment, 7*, 249-260.

Coogan, P.F., Adams, M., Geller, A.C., Brooks, D., Miller, D.R., Lew, R.A., et al. (1998). Factors associated with smoking among children and adolescents in Connecticut. *American Journal of Preventive Medicine, 15*(1), 17-24.

Crowley, T.J., MacDonald, M.J., & Walter, M.I.(1995). Behavioral anti-smoking trial in chronic pulmonary disease patients. *Psychopharmacology, 119*, 193-204.

Davis, W.E., and Zitsch, R.P. (1999). Statistics of Head and Neck Cancer. Health consequences of smoking. In S.E. Thawley, W.R. Panje, J.G. Batsakis, & R.D. Lindberg (Eds.*), Comprehensive Management of Head and Neck Tumor* (pp. 305-331). Philadelphia: W.B. Saunders Company.

Day, G.L., Blot, W.J., Shore, R.E., McLaughlin, J.K., Austin, D.F., Greenberg, R.S., et al. (1994). Second cancers following oral and pharyngeal cancers: Role of tobacco and alcohol. *Journal of the National Cancer Institute, 86*(2), 131-137.

Deleyiannis, F.W-B., Thomas, D.B., Vaughan, T.L., & Davis, S. (1996). Alcoholism: Independent predictor of survival with patients with head and neck cancer. *Journal of the National Cancer Institute, 88*(8), 542-549.

Derogatis, L.R., Morrow, G.R., Tetting, J., Penman, D., Piaselsky, S., Schmael, A.M., et al. (1983). The prevalence of psychiatric disorders among cancer patients. *Journal of the American Medical Association, 249*(6), 751-757.

Devins, G.M. & Edwards, P.J. (1988). Self-efficacy and smoking reduction in chronic pulmonary disease. *Behavior Research Therapy, 26*(2), 127-135.

DiClemente, Prochaska, & Gilbertini (1985). Self-efficacy and stages of self-change in smoking. *Cognitive Therapy and Research, 9*(2), 181-200.

Doherty, K., Kinnunen, T., Militello, F.S., & Garvey, A.J. (1995). Urges to smoke during the first month of abstinence: Relationship to relapse and predictors. *Psychopharmacology, 119*, 171-178.

Dudley, D.L. & Sitzman, J. (1988). Psychobiological evaluation and treatment of COPD. In A.J. McSweeney & I. Grant (Eds.). *Chronic Obstructive Pulmonary Disease: A Behavioral Perspective.* New York: 1988, PP. 183-235.

Duffy, S.A., Terrell, J.E., Valenstein, M., Ronis, D.L., Copeland, L.A., & Connors, M. (2002). Effect of smoking, alcoh9ol, and depression on the quality of life of head and neck cancer patients. *General Hospital Psychiatry, 24*, 140-147.

Duncan, C.L., Cummings, S.R., Hudes, E.S., Xahnd, E. & Coates, T.J. (1992). Quitting smoking: Reasons for quitting and predictors of cessation among medical patients. *Journal of General Internal Medicine, 7*, 398-404.

D'Zurilla, T.J., Nezu, A.M., & Maydeu-Olivares, A. (2002). *Manual for the social problem-solving inventory – revised (SPSI-R*). North Tonawanda, NY: Multi-Health Systems.

D'Zurilla, T.J. & Nezu, A.M. (1990). Development and preliminary evaluation of the social problem-solving inventory. *Psychological Assessment, 2*(2), 156-163.

D'Zurilla, TJ, Nezu, A.M., & Maydeu-Olivares, A. (In press). What is social problem solving? Meaning, models, and measures. In E. Chang, T. D'Zurilla, and L. Sanna (Eds.). *Social problem solving: Theory, research, and training*. Washington DC: American Psychological Association.

Enright, P.L. , Lebowitz, M.D., & Cockroft, D.W. (1994). Physiologic measures: Pulmonary function tests. *American Journal of Respiratory Critical Care, 149*, S9-S18.

Evans, R.I., Hansen, W.B., & Mittelmark, M.B. (1977). Increasing the validity of self-reports of smoking behavior on children. *Journal of Applied Psychology, 62*, 521-523.

Fabian, M.C., Irish, J.C., Brown, D.H., Liu, T.C., & Gullane, P.J. (1996). Tobacco, alcohol and oral cancer: The patient's perspective. *The Journal of Otolaryngology, 25*(2), 88-93.

Freund, K.M., D'Agostino, R.B., Belanger, A.J., Kannel., W.B., & Stokes, J. (1992). Predictors of smoking cessation: The Framingham Study. *American Journal of Epidemiology, 135*(9), 957-964.

Gail, M.H., Eegan, R.T., Feld, R., Ginsberg, R., Goodell, B., Hill, L., et al. (1984). The lung study group: Prognostic factors in patients with respected Stage I non-small cell lung cancer. A report form the Lung Cancer Study Group. *Cancer, 54*, 1802-1813.

Garfinkel, L. (1997). Trends in cigarette smoking in the United States. *Preventive Medicine, 26*, 447-450.

Gilbert, D.G., McClernon, F.J., Rabinovich, N.E., Plath, L.C., Jensen, R.A., & Meliska, C.J. (1997). Effects of smoking abstinence on mood and craving in men: Influences of negative affect-related personality traits, habitual nicotine intake, and repeated measurements. *Personality and Individual Differences, 25*, 399-423.

Gilbert, D.G., McClernon, F.J., Rabinovich, N.E., Plath., L.C., Masson, C.L., Anderson, A.E. et al. (2002). Mood disturbance fails to resolve across 31 days of cigarette abstinence in women. *Journal of Consulting and Clinical Psychology, 70*(1), 142-152.

Ginsberg, D., Hall, S.M., Reus, V.I., & Munoz, R.F. (1995). Mood and depression diagnosis in smoking cessation. *Experimental and Clinical Psychopharmacology, 3*(4), 389-395.

Ginseberg, JP, Klesges, RC, Johnson, KC, Eck, LH, Meyers, AW, & Winders, SA. (1997). The relationship between a history of depression and adherence to a multicomponent smoking-cessation program. *Addictive Behaviors, 22*(6), 783-787.

Glassman, A.H. (1993). Cigarette smoking: Implications for psychiatric illness. American Journal of Psychiatry, 150(4), 546-553.

Glynn, S.M., Gruder, C.L., & Jegerski, J.A. (1986). Effects of biochemical validation of self-reported cigarette smoking on treatment success and on misreporting abstinence. *Health Psychology, 5*(2), 125-136.

Greden, J.F., & Pomerlau, O. Caffeine-related disorders and nicotine-related disorders. (2001). In H.I. Kaplan and B.J. Saddock (Eds.), *Comprehensive Textbook of Psychiatry/VI,* (pp. 799-810). Baltimore: Williams & Wilkins.

Green, S.B. (1995a). Community intervention trial for smoking cessation (COMMIT): I. Cohort results from a four-year community intervention. American Journal of Public Health, 85(2), 183-192.

Green, S.B. (1995b). Community intervention trial for smoking cessation (COMMIT):II. Changes in adult cigarette smoking prevalence. *American Journal of Public Health, 85*(2), 193-200.

Greenberg, G.D., Ryan, J.J., & Bourlier, P.F. (1985). Psychological and neuropsychologic aspects of COPD. *Psychosomatics, 26*(1), 29-33.

Gritz, E.R., Carr, C.R., Rapkin, D., Abemeyer, E., & Chang, L-J.C., Wong, W.K., et al. (1993). Predictors of long-term smoking cessation in head and neck cancer patients. *Cancer Epidemiology, Biomarkers & Prevention, 2,* 261-270.

Gritz, E.R., Kristeller, J.L., & Burns, D.M. (1993). Treating nicotine addiction in high-risk groups and patients with medical co-morbidity. In C.T. Orleans & J. Slade (Eds.), *Nicotine Addiction: Principles and Management (*pp. 279-309). New York: Oxford University Press.

Gritz, E.R., Nisenbaum, R., Alashoff, R., & Holmes, E.C. (1991). Smoking behavior following diagnosis in patients with Stage I non-small cell lung cancer. *Cancer Causes Control, 2,* 105-112.

Grove, J.R. (1993). Attributional correlates of cessation self-efficacy among smokers. *Addictive Behaviors, 18,* 311-320.

Gunn (1983). Smoking clinic failures and recent life stress. *Addictive Behaviors, 8,* 83-87.

Hair, J.F., Anderson, R.E., Tatham, R.L., & Black, W.C. (1995). *Multivariate data analysis* (4th ed.). Upper Saddle River, NJ: Prentice Hall.

Haley, N.J., Axelrod, C.M., & Tilton, K.A. (1983). Validity of self-reported smoking behavior: Biochemical analyses of cotinine and thiocyanate. *American Journal of Public Health, 73*(10), 1204-1207.

Hall, S.M., Munoz, R.F., & Reus, V.I. (1994). Cognitive-behavioral intervention increases abstinence rates for depressive-history smokers. *Journal of Clinical and Consulting Psychology, 62*(1), 141-146.

Hall, S.M., Munoz, R.F., Reus, V.I., & Sees, K.L. (1993). Nicotine, negative affect, and depression. *Journal of Consulting and Clinical Psychology, 61*(5), 761-767.

Hall, S.M., Tunstall, C., Rugg, D., Jones, R.T., & Benowtiz, N. (1985). Nicotine gum and behavioral treatment in smoking cessation. *Journal of Consulting and Clinical Psychology, 53*(2), 256-258.

Hecht, J.P., Emmons, K.M., Brown, R.A., Everett, K.D., Farrell, N.C., Hitchcock, P., & Sales, S.D. (1994). Smoking interventions for patients with cancer: Guidelines for nursing practice. *Oncology Nursing Forum, 21*(10), 1657 – 1666.

Helsper, J.T., & Dollinger, M. (1991). Head and neck. In M. Dollinger, E.H. Rosenbaum, & G. Cable, (Eds.). *Everyone's guide to cancer therapy*. (pp. 410-430). Kansas City, MO: Andrews and McMeel.

Heppner, PP, Kampa, M., & Brunning, L. (1987). The relationship between problem-solving self-appraisal and indices of physical and psychological health. *Cognitive Therapy and Research, 11*(2), 155-168.

Hinds, M.W., Yang, H., Stemmermann, G., Lee, J., & Kolonel, L.N. (1982). Smoking history and lung cancer survival in women. *Journal of the National Cancer Institute, 68*(3), 395-399.

Holland, J.F. (1998). Biology of cancer for the psycho-oncologist. In J.C. Holland (Ed.). *Psycho-oncology*. New York: Oxford University Press.

Horowitz, M.B., Hindi-Alexander, M., & Wagner, T.J. (1985). Psychological mediators of abstinence, relapse, and continued smoking: A one-year follow-up of a minimal intervention. *Addictive Behaviors, 10*, 29-39.

Janz, N.K. & Becker, M.H. (1984). The health belief model: A decade later. *Health Education Quarterly, 11*(1), 1-47.

Johnston, W.D., & Ballantyne, A.J. (1977). Prognostic effect of tobacco and alcohol use in patients with oral tongue cancer. *The American Journal of Surgery, 134*,

444-447.

Johnston-Early, A., Cohen, M.H., Minna, J.D., Paxton, L.M., Fossick, B.E., Ihde, D.C. et at al. (1980). *JAMA, 244*, 2175-2179.

Jones, E.E. & Sigall, H. (1971). The bogus pipeline: A new paradigm for measuring affect and attitude. *Psychological Bulletin, 76*(5), 349-364.

Kaplan, R.M., Reis, A.L., Prewitt, L.M., & Eakin, E. (1994). Self-efficacy expectations predict survival for patients with chronic obstructive pulmonary disease. *Health Psychology, 13*(4), 366-368.

Kick, S.D. & Cooley, D.D. (1997). Depressive, not anxiety, symptoms are associated with current cigarette smoking among university internal medical patients. *Psychosomatics, 38*, 132-139.

Kowalski, S.D. (1997). Self-esteem and self-efficacy as predictors of success in smoking cessation. *Journal of Holistic Nursing, 15*(2), 128-142.

Kviz, F.J., Clark, M.A., Crittenden, K.S., Freels, S. & Warnecke, R.B. (1994). Age and readiness to quit smoking. *Preventive Medicine, 23*, 211-222.

Labott, S.M. (1998). COPD and other respiratory diseases. In P.M. Carnic and S.J. Knight (Eds.). *Clinical Handbook of Health Psychology: A Practical guide to effective interventions* (pp. 410-430). Seattle: Hogrefe & Huber Publications.

Lawrence, & Rubinson (1986). Self-efficacy as a predictor of smoking behavior in young adolescents. *Addictive Behaviors, 11*, 367-382.

Lee, C. (1989). Perceptions of immunity to disease in adult smokers. *Journal of Behavioral Medicine, 12*(3), 267-277.

Leidy, N.K. & Traver, G.A. (1995). Psychophysiologic factors contributing to functional performance in people with COPD: Are there gender differences. *Research in Nursing Forum, 18*, 535-546.

Lev, E.L. (1997). Bandura's theory of self-efficacy: Applications to oncology. *Scholarly Inquiry for Nursing Practice: An International Journal, 11*(1), 21-37.

Lewin, F., Norell, S.E., Johanssen, H., Gustavsson, P., Weinberg, J., Bioklund, A., & Rutqvist, L.E., (1998). Smoking tobacco. Oral snuff, and alcohol in the etiology of squamous cell carcinoma of the head and neck. *Cancer, 82*(7), 1367-1375.

Lichtenstein, E., Lando, H.A., & Nothwehr, F. (1994). Readiness to quit as a predictor of smoking changes in the Minnesota Heart Health Program. *Health Psychology, 13*, 393-336.

Light, R.W., Merrill, E.J., Despars, J.A., Gordon, G.H., & Mutalipassi, L.R. (1985). Prevalence of depression and anxiety in patients with COPD: Relationship to functional capacity. *Chest, 87*(1), 35-38.

Lillington, G.A., Leonard, C.T., & Sachs, D.P.L. (2000). Smoking cessation: Techniques and benefits. *Clinics in Chest Medicine, 21*(1), 199-207.

Massie, M.J. & Popkin, M.K. Depressive disorders. (1998). In J.C. Holland (Ed.). *Psycho-oncology*, 518-540. Oxford University Press: New York.

Matheny, K.B & Weatherman, K.E. (1998). Predictors of smoking cessation and maintenance. *Journal of Clinical Psychology, 54*(2), 223-235.

Mayo Foundation Nicotine Dependence Center Patient Questionnaire. In C.T. Orleans & J. Slade (Eds.). *Nicotine addiction*. New York: Oxford University Press.

McCoy, S.B., Gibbons, F.X., Reis, T.J., Gerrard, M., Luus, C.A.E., & Sufka, A.V.W. (1992). Perceptions of smoking risk as a function of smoking status. *Journal of Behavioral Medicine, 15*(5), 469-488.

McIntyre-Kingsolver, K., Lichtnestein, E. & Mermelstein, R.J. (1986). Spouse training in a multicomponent smoking0cessatipon program. *Behavior Therapy, 17*, 67-74.

McKusker, K. (1992). Mechanisms of respiratory tissue and injury from cigarette smoking. *The American Journal of Medicine,* 93 (Supplement 1A), 18-21.

McNair, D.M., Lorr, M. & Droppleman, L.F. (1992). *Manual for the Profile of Mood*

States. San Diego, CA: EdITS.

McWhorter, W.P., Boyd, G.M., & Mattson, M.E. (1990). Predictors of quitting smoking: The NHANES I follow-up experience. *Journal of Clinical Epidemiology, 46,* 1399-1405.

Moadel, A.B., Ostroff, J.S., & Schantz, S.P. (1998). Head and Neck Cancer. In J.C. Holland (Ed.). *Psycho-oncology,* 314-323. Oxford University Press: New York.

Molloy, G.N., Wolstencroft, K., King, N.J., Lowe, A.R., Gardner, P.L. & Rowley, G.L. (1997). Locus of control of smokers, nonsmokers, and nonpracticing smokers. *Psychological Reports, 81*, 781-782.

Moore, C. (1971). Cigarette smoking and cancer of the mouth, pharynx, and larynx. *The Journal of the American Medical Association, 218*(4), 553-558.

Mothersill, K.J., McDowell, I., & Rosser, W. (1988). Subject characteristics and long-term post-program smoking cessation. *Addictive Behaviors, 13*(1), 29-36.

Muller, M.M. (1992). Do smokers differ form nonsmokers in anger and anger coping? The investigation of a more specific trait in the search for a 'smokers personality.'" *Personality and Individual Differences, 13*(3), 281-285.

Murray, D.M. & Perry, C.L. (1987). The measurement of substance use among adolescents: When is the "bogus pipeline" method needed? *Addictive Behaviors, 12,* 225-233.

Murray, R.P., Anthonisen, N.R., Connett, J.E., Wise, R.A., Lindgren, PG., Greene, P.G. et al. (1998). Effects o multiple attempts to quit smoking and relapses to smoking on pulmonary function. *Journal of Clinical Epidemiology,* 51(12), 1317-1326.

Murray, R.P., Connett, J.E., Lauger, G.G., & Voelker, H.T. (1993). Error in smoking measures: Effects of Intervention on relations of cotinine and carbon monoxide to self-reported smoking. *American Journal of Public Health, 83*(9), 1251-1257.

Murray, R.P., Istvan, J.A., Voelker, H.J., Rigdon, M.A., & Wallace, M.D. (1995).

Level of involvement with alcohol and success at smoking cessation in The Lung Health Study. *Journal of Studies on Alcohol, 56*, 74-82.

Murray, D.M., O'Connell, C.M., Schmid, L.A. & Perry, C.L. (1987). The validity of smoking self-reports buy adolescents: A reexamination of the bogus pipelines procedure. *Addictive Behaviors, 12*, 7-15.

National Center for Health Statistics (1998). *Current estimates from the National Health Interview Survey,* United States, selected years, 1971-1994.

Newcomb, P.A. & Carbone, P.P. (1992). The health consequences of smoking – Cancer. *Medical Clinics of North America, 76*(2), 305-331.

Nezu, A.M. (1985). Differences in psychological distress between effective and ineffective problem solvers. *Journal of Counseling Psychology, 32*(1), 135-138.

Nezu, A.M. (1986a). Negative life stress and anxiety: Problem solving and as a moderating variable. *Psychological Reports, 58*, 279-283.

Nezu, A.M. (1986b). Efficacy of a social problem-solving therapy approach for unipolar depression. *Journal of Consulting and Clinical Psychology, 54*(2), 196-202.

Nezu, A.M. (1987). A problem-solving formulation of depression: A literature review and proposal of a pluralistic model. *Clinical Psychology Review, 7*, 121-144.

Nezu, A.M. & D'Zurilla, T.J. (1989). Social Problem Solving and Negative Affective Conditions. In PC Kendal and D. Watson (Eds.). (1989). *Anxiety and depression: Distinctive and overlapping features*. New York: Academic Press.

Nezu, A.M., Kalmar, K., Ronan, G.F., & Clavijo, A. (1986). Attributional correlates of depression: An interactional model including problem solving. *Behavior Therapy, 17*, 50-56.

Nezu, A.M., Nezu, C.M., Friedman, S.H., Faddis, S, & Houts, P.S. (1998). *Helping cancer patients cope: A problem-solving approach*. Washington D.C.: American Psychological Association.

Nezu, C.M., Nezu, A.M., Friedman, S.H., Houts, P.S., DelliCarpini, L.A., Bildner, C. & Faddis, S. (1999). Cancer and psychological distress: Two investigations regarding the role of social problem-solving. *Journal of Psychosocial Oncology, 16*(3/4), 27-40.

Nezu, A.M., Nezu, C.M., Friedman, S.H., Houts, P.S., & Faddis, S. (1999). Project Genesis: Application of problem-solving therapy to individuals with cancer. *The Behavior Therapist, 20*(9), 155-158.

Nezu, A.M., Nezu, C.M., Houts, P.S., Friedman, S.H., & Faddis, S. (1999). Relevance of problem-solving therapy to psychosocial oncology. *Journal of Psychosocial Oncology, 16*(3-4), 5-26.

Nezu, A.M., Nezu, C.M., McClure, K.S., & Zwick, M.L. (2002). Assessments of depression. In I.H. Gotlib (Ed.). *Handbook of depression* (pp. 61-85). New York: Guilford Press.

Nezu, A.M., Nezu, C.M., & Perri, M.G. (1989). Social problem-solving therapy for unipolar depression: An initial dismantling investigation. *Journal of Consulting and Clinical Psychology, 57*(3), 408-413.

Nezu, A.M., Nezu, C.M., Rothenberg, J.L., & D'Zurilla, T.J. Problem-Solving Therapy. In J.S. Kantor (Ed.). (1996). *Clinical depression during addiction recovery: Prognosis, diagnosis, and treatment*. New York: Marcel Dekker, Inc.

Nezu, A.M. & Ronan, G.F. (1985). Life stress, current problems, problem solving, and depressive symptoms: An integrative model. *Journal of Consulting and Clinical Psychology, 53*(5), 693-697.

Nezu, A.M. & Ronan, G.F. (1988). Social problem solving as a moderator of stress related depressive symptoms: A prospective analysis. *Journal of Counseling Psychology, 35*(2), 134-138.

Nezu, A.M., Nezu, C.M., McClure, K.S., & Zwick, M. L. (2002). Assessments of depression. In I.H. Gotlib and C.L. Hammen (Eds.). *Handbook of Depression* (pp. 61-85). New York: Guilford Press.

Ockene, J.K., Benfari, R.C., Nuttall, R.L., Hurwitz, I., & Ockene, I.S. (1982).

Relationship of psychosocial factors to smoking behaviors change in an intervention program. *Preventive Medicine, 11*(1), 13-28.

O'Connor, E.A., Carbonari, J.P., & DiClemente, C.C. (1996). Gender and smoking cessation: A factor structure comparison of processes of change. *Journal of Consulting and Clinical Psychology, 64*(1), 130-138.

O'Leary, A (1985). Self-efficacy and health. *Behavioral Research and Therapy, 23*, 437-452.

Orleans, C.T., Kristeller, J.L, & Gritz, E.R. (1993). Helping hospitalized smokers quit: New directions for treatment and research. *Journal of Consulting and Clinical Psychology, 61(*5), 778-789.

Osler, M., Prescott, E., Gidtfredsen, N., Hein, H.O. and Schnohr, P. (1999). Gender and determinants of smoking cessation: A longitudinal study. *Preventive Medicine, 29*, 57-62.

Ossip-Klein, D.J., Giovino, G.A., Megahed, N., Black, P.M., Emont, S.L., Stiggins, J., et al. (1991). Effects of a smokers' hotline: Results of a 10-county self-help trial. *Journal of Consulting and Clinical Psychology, 59*, 325-332.

Ostroff, J.S., Jacobsen, P.B., Moadel, A.B., Spiro, R.H., Shah, J.P., Strong, E.W., et al. (1995). Prevalence and predictors of continued tobacco use after treatment of patients with head and neck cancer. *Cancer, 75*(2), 569-576.

Owen, N. & Brown, S.L. (1991). Smokers unlikely to quit. *Journal of Behavioral Medicine, 14*(6), 627-636.

Pederson, L.L., Baskerville, J.C., & Wanklin, J.M. (1982). Multivariate statistical models for predicting change in smoking behavior following physician advice to quit smoking. *Preventive Medicine, 11*, 536-549.

Pederson, L.L., Wanklin, J.M., & Baskerville, J.C. (1984). The role of health beliefs in compliance with physician advice to quit smoking. *Social Science Medicine, 19*(5), 573-580.

Pederson, LL, Wanklin, JM, & Lefcoe, NM (1991). The effects of counseling on smoking cessation among patients hospitalized with chronic obstructive

pulmonary disease: A randomized clinical trial. *The International Journal of the Addictions, 26*(1), 107-119.

Pederson, L.L., Williams, J.I, & Lefcoe, N.M. (1980). Smoking cessation among pulmonary patients as related to type of respiratory disease and demographic variables. *Canadian Journal of Public Health, 71*, 191-194.

Petitti, D.B., Friedman, G.D., & Kahn, W. (1981). Accuracy of information on smoking habits provided on self-administered research questionnaires. *American Journal of Public Health, 71*, 308-311.

Petty, T.L. (1988). Medical management of COPD. In A.J. McSweeny & I. Grant (Eds.). *Chronic obstructive pulmonary disease: A behavioral perspective.*(pp. 87-103). New York: Marcel Dekker, Inc.

Pirie, P.L., Murray, D.M., & Luepker, R.V. (1991). Gender differences in cigarette smoking and quitting in a cohort of young adults. *American Journal of Public Health, 81*, 324-327.

Postma, D.S. & Sluiter, H.J. (1989). Prognosis of chronic obstructive pulmonary disease: The Dutch experience. *American Review of Respiratory Disease, 140*(3 pt 2), S100-S105.

Prochaska, J.O. & DiClemente, C.C. (1983). Stages and process of self-change of smoking: Toward an integrative model of change. *Journal of Consulting and Clinical Psychology, 51*, 390-395.

Prochaska, J.O., DiClemente, C.C., & Norcross, J.C. (1992). In search of how people change: Applications to the addictive behaviors. *American Psychologist, 47*(9), 1102-1114.

Prochaska, J.O., Redding, C.A., & Evers, K.E. (1997). The transtheoretical model and stages of change. In *Health Behavior and Health Education: Theory, research, and practice*. K. Glanz, F.M. Lewis, and B.K. Rimer (eds). Jossey-Bass Publishers: San Francisco.

Prochaska, J.O., Velicer, V.F., Rossi, J.S., Goldstein, M.G., Marcus, B.H., Rakowski, W., et al. (1994). Stages of change and decisional balance of twelve problem behaviors. *Health Psychology, 13*, 39-46.

Pussik, S.D., Dugan, W., McDonald, M.V., Rosenfeld, B., Theobold, D.E., & Edgerton, S. (1998). Oncologist's recognition of depression in their patients with cancer. *Journal of Clinical Oncology, 16*(4), 1594-1600.

Pederson, L.L., Wanklin, J.M., & Lefcoe, N.M. (1991). The effects of counseling on smoking cessation among patients hospitalized with chronic obstructive pulmonary disease: A randomized clinical trial. *The International Journal of the Addictions, 26*(1), 107-119.

Postma, D.S., & Sluiter, H.J. (1989). Prognosis of chronic obstructive pulmonary disease: The Dutch experience. *American Review of Respiratory Disease, 140*, S100-S105.

Rabois, D. & Haaga, D.A.F. (1997). Cognitive coping, history of depression, and cigarette smoking. *Addictive Behaviors, 22*(6), 789-796.

Rosenstock, I.M., Strecher, V.J., & Becker, M.H. (1988). Social learning theory and the health belief model. *Health Education Quarterly, 15*(2), 175-83.

Royce, J.M., Corbett, K., Sorensen, G., & Ockene, J. (1997). Gender, social pressure and smoking cessations: The community intervention trial for smoking cessation (COMMIT) at baseline. *Social Science Medicine, 44*, 359-370.

Salive, M.E., Cornoni-Huntley, J., LaCroix, A.Z., Ostfeld, A.M., Wallae, R.B. & Hennekens, C.H. (1992). Predictors of smoking cessation and relapse in older adults. *American Journal of Public Health, 82*(9), 1268-1271.

Sarna, L. (1995). Smoking behaviors of women after diagnosis with lung cancer. *IMAGE: Journal of Nursing Scholarship, 27*(1), 35-41.

Schnoll, R.A., Malstrom, M., Randi, C.J., Rothman, R.L., Miller, S.M., Ridge, J.A., et al. (2002). Correlates of tobacco use among smokers and recent quitters diagnosed with cancer. *Patient Education and Counseling, 46*, 137-145.

Sethi, J.M. & Rochester, C.L. (2000). Smoking and chronic obstructive pulmonary disease. *Clinics in Chest Medicine, 21*(1), 67-86.

Shiffman, S. (1986). A cluster-analytic classification of smoking relapse episodes. *Addictive Behaviors, 11,* 295-307.

Shiffman, S., Hickcox, M., Paty, J.A., Gnys, M., Kassel, J.D., & Richards, T.J. (1996). Progression from a smoking lapse to relapse: Prediction from abstinence violation effects, nicotine dependence, and lapse characteristics. *Journal of Consulting and Clinical Psychology, 64*(5), 993-1002.

Shiffman, S., Hickcox, M., Pay, J.A., Gnys, M., Richards, T., & Kassel, J.D., (1997). Individual differences in the context of smoking lapse episodes. *Addictive Behaviors, 22*(6), 797-811.

Shiffman, S., Shumaker, S.A., Abrams, D.B., Cohen, S., Garvey, A., Grunberg, N.E., et al. (1986). Task force 2: Models of smoking relapse. *Health Psychology, 5*(Suppl.), 13-27.

Sillett, R.W., Wilson, M.B., Malcolm, R.E., & Bal K.P. (1978). Deception among smokers. *British Medical Journal, 2* (6146), 1185-1186.

Silverman, S., Greenspan, D. & Gorsky, M. (1983). Tobacco usage in patients with head and neck carcinomas: A follow-up study on habit changes and second primary oral/oropharyngeal cancers. *Journal of the American Dental Association, 106,* 33-35.

Spielberger, C.D., Foreyt, J.P., Reheiser, E.C., & Poston, W.S.C. (1998). Motivational, emotional, and personality characteristics of smokeless tobacco users compared with cigarette smokers. *Personality and Individual Differences, 25,* 821-832.

Spitz, M.R., Fueger, M.P., Chamberlain, R.M., Goepfert, H., & Newell, G.R. (1990). Cigarette smoking patterns after treatment of upper aeordigestive tract cancers. *Journal of Cancer Education, 5,* 109-113.

Spitz, M.R., Fueger, M.P., Eriksen, P, & Newell, G.R. (1990). Cigarette smoking patterns of cancer patients. *Advances in Cancer Control: Screening and Prevention Research,* 73-82.

Stevens, M.H., Gardner, J.W., Parkin, J.L., & Johnson, L.P. (1983). Head and neck cancer survival and life-style change. *Archives of Otolaryngology, 109,* 746-749.

Stookey, G.K., Katz, B.P., Olson, B.L., Drook, C.A., & Cohen, S.J. (1987). Evaluation of biochemical validation measures in determination of smoking status. *Journal of Dental Residency, 66*(10), 1597-1601.

Strecher, V.J., Kreuter, M.W., & Kobrin, S.C. (1995). Do cigarette smokers have unrealistic perceptions of their heart attack, cancer and stroke risks? *Journal of Behavioral Medicine, 18*(1), 45-45-54.

Strecher, V.J. & Rosenstock, I.M. (1997). The health belief model. In *Health Behavior and Health Education: Theory, research, and practice*. K. Glanz, F.M. Lewis, and B.K. Rimer (Eds). Jossey-Bass Publishers: San Francisco.

Stronks, K, Van de Mheen, H.D., Looman, C.W.N., & Mackenback, J.P. (1997). Cultural, material, and psychological correlates of the socioeconomic gradient in smoking behavior among adults. *Preventive Medicine, 26*, 754-766.

Swan, G.E., Ward, M.M., & Jack, L.M. (1996). Abstinence effects as predictors of 28-day relapse in smokers. *Addictive Behaviors, 21*(4), 481-490.

Takemura, Y., Akanuma, M., Kikuchi, S., and Inaba, Y. (1999). Cross-sectional study on the relationship between smoking or smoking cessation and trait anxiety. *Preventive Medicine, 29*, 496-500.

Talamini, R., La Vecchia, C., Levi, F., Conti, E., Favero, A. & Franceschi, S. (1998). Cancer of the oral cavity and pharynx in nonsmokers who drink alcohol and in nondrinkers who smoke tobacco. *Journal of the National Cancer Institute, 90*, 1901-1903.

Tate, J.C., Stanton, A.L., Green, S.B., & Schmitz, J.M. (1996). Assessing the validity of nicotine abstinence effects by self- and observer ratings under "blinded" conditions. *Experimental and Clinical Psychopharmacology, 4*(3), 330-335.

Taylor, D.H., Hasselblad, V., Henley, S.J., Thun, M.J., & Sloan, F. (2002). Benefits of smoking cessation for longevity. *American Journal of Public Health, 92*(6), 990-996.

Tessaro, I, Lyna, P.R., Rimer, B.K., Heisler, J., Woods-Powell, C.T., Yarnall, K.S.H., et al. (1997). Readiness to change smoking behavior in a community health center population. *Journal of Community Health, 22*(1), 15-31.

Thun, M.J., Day-Lally, C., & Myers, D.G. (1995). Trends in tobacco smoking and mortality form cigarette use in cancer prevention studies I (1959-1965) and II

(1982-1988). *American Journal of Public Health, 85*, 1223-1229.

Tsuda, A., Steptoe, A., West, R., Fieldman, G., & Kirschbaum, C. (1996). Cigarette smoking and psychophysiological stress responsiveness: Effectiveness of recent smoking and temporary abstinence. *Psychopharmacologia, 126*(3), 226-233.

U.S. Department of Health and Human Services. (1988). *The health consequences of smoking; Nicotine Addiction. A Report of the Surgeon General*. DHHS Publication No. (CDC) 88-8406, 563-587.

U.S. Department of Health and Human Services. (1984). *The health consequences of smoking: Chronic obstructive lung disease. A Report of the Surgeon General*. (DHHS Publication No. 84-50205). Washington DC: Author.

Utz, S.W., Shuster, G.F., Merwin, E., & Williams, B. (1994). A community-based smoking-cessation program: Self-care behaviors and success. *Public Health Nursing, 11*, 291-299.

Vander Ark, W., DiNardo, L.J., & Oliver, D.S. (1997). Factors affecting smoking cessation in patients with head and neck cancer. *Laryngoscope, 107*, 888-892.

Velicer, W.E., Prochaska, J.O., Rossi, J.S., & Snow, M.G. (1992). Assessing outcome in smoking cessation studies. *Psychological Bulletin, 111*(1), 23-41.

Vogt, T.M., Selvin, S., Widdowson, G., & Hulley, S.B. (1977). Expired air carbon monoxide and serum thiocyanate as objective measures of cigarette exposure. American *Journal of Public Health, 67*(6), 545-549.

Wang, M.Q., Fitzhugh, E.C., Eddy, J.M., & Westerfield, R.C. (1998). School dropouts' attitudes and beliefs about smoking. *Psychological Reports, 82*, 984-986.

Wang, M.Q., Fitzhugh, E.C., Westerfield, R.C., & Eddy, J.M. (1994). Predicting smoking status by symptoms of depression for U.S. adolescents. *Psychological Reports,75*, 911-914.

Ward, K.D., Klesges, R.S., Zbikowski, N., Blis, R.E., & Garvey, A.J. (1997). Gender differences in the outcomes of unaided smoking cessation attempt. *Addictive Behaviors, 22*, 521-5433.

Weaver, T.E., & Narsavage, G.L. (1992). Physiological and psychological variables

related to functional states in chronic obstructive pulmonary disease. *Nursing Research, 41*(5), 286-291.

Weinberger, M., Greene, Y.J., Mamlin, J.J., & Jerin, M.J. (1981). Health beliefs and smoking behavior. *American Journal of Public Health, 71*(11), 1253-1255.

Wewers, M.E. (1988). The role of postcessation factors in tobacco abstinence: Stressful events and coping responses. *Addictive Behaviors, 13*, 297-302.

Wise, R.A. (1997). Changing smoking patterns and mortality from chronic obstructive pulmonary disease. *Preventive Medicine, 26*, 418-421.

Wise, R.A., Szklo, M., Matanoski, G. & Neugut, A. (1997). Workshop discussion panel III: Implications of the changing tobacco-related mortality from COPD in the CPS-1 and CPS-2 surveys. *Preventive Medicine, 26*, 457-459.

Witt, E.M., Kaelin, J.A., & Stoner, S.B. (1988). Smoking behavior and anger. *Psychological Reports, 63*, 117-118.

Wynder., E.L., Dodo, H., Bloch, D.A., Gantt, R., & Moore, O.S. (1969). Epidemiologic investigation of multiple primary cancer of the upper alimentary and respiratory tracts. *Cancer, 24*(4), 730-739.

Xu, X., Dockery, D.W., Ware, J.H., Speizer, F.E., & Ferris Jr., B.G. (1992). Effects of cigarette smoking on rates of loss of pulmonary function in adults: A longitudinal assessment. *American Review of Respiratory Disease, 146*, 1245-1348.

Yates, A.J. & Thain, J. (1985). Self-efficacy as a predictor of relapse following voluntary cessation of smoking. *Addictive Behaviors, 10*, 291-298.

Zvolensky, M.J., Feldner, M.T., Eifert, G.H., and Brown, R.A. (2001). Affective style among smokers: Understanding anxiety sensitivity, emotional reactivity, and distress tolerance using biochemical challenge. *Addictive Behaviors, 26*, 901-915.

APPENDIX A: Demographic Form

Smoking, Distress, and Problem Solving

Clinic: Hayden Hahnemann Pulmonary MCP Pulmonary

Temple head and Neck Temple Pulmonary

Doctor: _____

I. Demographic Information

Subject Number: _____ Date: _____

Name: _____

Address: _____

_____

_____

Telephone Number: Home (_____) _____-_____

Work (_____)_____ - _____

Best time and place to contact you: Time: _____ Place: Home

Work

Okay to leave a message? Yes No

Gender: M F

Age: _____ Date of Birth: _____

Ethnicity:

African American Asian Caucasian

Hispanic Other: Please specify: _____

Marital Status:

Single Living with Significant Other If so, for how long: _____

Married If so, for how long: _____ Separated Month/Yr: _____

Divorced Month/Yr: _____ Widowed Month/Yr: _____

Number of Years of Education: _____ Highest Diploma/Degree: _____

Current Occupation: _____

If retired: 1) What was your occupation? _____ 2) Year you retired _____

Current Total Family Income: _____

II. Illness Variables

What is your current diagnosis?

_____

In what month and year were you first diagnosed?

_____

What is your current treatment?

_____

What treatment have you had in the past?

_____

Do you have any other medical conditions? If so, please list ALL other medical conditions and medications.

III. Smoking Variables

Please Note: Your honesty regarding these questions is very important to us and the research. Your answers to these questions will not be revealed to your physicians and other individuals on your health care team.

If you are a **current** smoker, please complete the questions in Part A.

If you have smoked at least one puff of a cigarette in the past 7 days, then please begin at Part A.

If you are a **former** smokers, please complete questions in Part B.

If you have not smoked even one cigarette in the last 7 days, please begin at Part B.

Part A (Current Smoker)

How old were you when you smoked your first cigarette? _____

How old were you when you first started regular daily cigarette smoking? _____

At the time you were first diagnosed, how many cigarettes, on average, did you smoke per day? _____

On average, how many cigarettes are you *currently* smoking per day? _____

Over the past six months, how many cigarettes, *on average*, did you smoke per day?

_____

When your smoking is/was the heaviest, how many cigarettes do you smoke per day?

_____

On average *of the entire time you have smoked*, how many cigarettes did you/have you smoke per day?

_____

Do you inhale? Always Sometimes Never

Please check the appropriate boxes:

	Never	Past Only	Currently
Smoke a pipe?			
Smoke cigars?			
Chew snuff?			
Chew tobacco?			

When do you smoke the heaviest (check one box only):

Mornings Afternoons Evenings Throughout the day

How soon after you wake up do you smoke your first cigarette?
____ immediately

____ within 30 minutes

____ between 30 minutes and one hour

____ beyond one hour

Which cigarette would be the most difficult to give up?

First cigarette in the morning

After meals

During or after stressful situations

During social occasions

In what situations do you smoke?

In public

At work

At home

In the presence of certain relatives

Do you have children? In the presence of them

At meetings

Inside the home of non-smokers

Do you have a car? In your car when non-smokers are with me

In other peoples cars

In restaurants

Other. Please specify _____

Have you ever tried to cut down or limit your smoking? Yes No

How many times? How many times have you *attempted* to stop smoking? _____

How many times have you stopped smoking *for at least one day*? _____

What is the longest time you have ever stopped smoking? _____

How long was this? Less than a day
At least one day but less than a week
At lease one week but less than a month
At least one month but less than one year
One year or more

What methods did you use to quit smoking? (e.g. "cold turkey," patch, gum, Zyban; support group): _____

What symptoms did you experience when you stopped smoking in the past? (Please check all that apply):

Craving Anxiety Restlessness
Decreased heart rate Increased eating Difficulty concentrating
Irritability Other: _____

Do you believe that quitting smoking would be beneficial to your health?

Yes No Not sure

Who lives in your household? _____

How many of these individuals are current smokers and what is their relationship to you?

_____

How many days per week, on average, do you drink alcoholic beverages? _____

On the days that you drink alcohol, how many drinks, on average, do you have? _____
(One drink = 8 ounces of beer (or) 4 ounces of wine (or) 1.5 ounces of liquor)

Part B. (Non Smoker)
How old were you when you smoked your first cigarette? _____

How old were you when you first started regular daily cigarette smoking?_____

At the time you were first diagnosed, how many cigarettes, on average, did you smoke per day? _____

On average, how many cigarettes did you smoke per day immediately before you quit?
_____

When your smoking was the heaviest, how many cigarettes did you smoke per day?
_____

On average of the entire time you have smoked, how many cigarettes did you smoke per day?
_____

Did you inhale? Always Sometimes Never

Please check the appropriate boxes:

	Never	Past Only	Currently
Smoke a pipe?			
Smoke cigars?			
Chew snuff?			
Chew tobacco?			

When did you smoke the heaviest (check one box only):

Mornings Afternoons Evenings

When was the last time you smoked a cigarette (month and year)? _____

When you were a smoker, how soon after you woke up did you smoke your first cigarette?

____ immediately

____ within 30 minutes

____ between 30 minutes and one hour

____ beyond one hour

Which cigarette was the most difficult to give up?

First cigarette in the morning

After meals

During or after stressful situations

During social occasions

In what situations did you smoke?

In public

At work

At home

Do you have children? In the presence of them

Inside the home of non-smokers

Do you have a car? In your car when non-smokers were with you

In other peoples cars

In restaurants

Other. Please specify _____

Prior to your final, successful quit attempt, had you ever *attempted* to quit smoking?

Yes No How many times? _____

Of those times, how many times had you stopped smoking for *at least one day*? _____

What is the longest time you have ever stopped smoking? _____

How long was this? Less than a day
At least one day but less than a week
At lease one week but less than a month
At least one month but less than one year
One year or more

What methods have you used in the (past or currently) to stop smoking? (e.g. "cold turkey," patch, gum, Zyban; support group): _____

What symptoms did you experience when you stopped smoking? (Please check all that apply):

Craving Anxiety Restlessness
Decreased heart rate Increased eating Difficulty concentrating
Irritability Other: _____

For assessor:

Mark this box if difficult to assess because quit upon diagnosis and surgery:

Do you believe that quitting smoking was beneficial to your health?

Yes No Not sure

Who lives in your household? _____

How many of these individuals are current smokers and what is their relationship to you?

_____

How many days per week, on average, do you drink alcoholic beverages? _____

On the days that you drink alcohol, how many drinks, on average, do you have? _____
(One drink = 8 ounces of beer (or) 4 ounces of wine (or) 1.5 ounces of liquor)

APPENDIX B: Social Problem-Solving Inventory – Revised

Sample Test Items of the SPSI Scales

Positive Problem Orientation

#7 When my first efforts to solve a problem fail, I know if I persist and do not give up too easily, I will be able to eventually find a good solution.

Negative Problem Orientation

13 When I am faced with a difficult problem, I doubt that I will be able to solve it on my own no matter how hard I try.

Rational Problem Solving

#24 When making decisions, I consider both the immediate consequences and long-term consequences of each option.

#44 When I have a problem to solve, I examine what factors or circumstances in my environment might be contributing to the problem.

Impulsivity/Carelessness

#34 When I have a decision to make, I do **not** take the time to consider the pros and cons of each option.

Avoidance Style

#23 I prefer to avoid thinking about the problems in my life instead of trying to solve them.

APPENDIX C: Profile of Mood States

Sample Test Items

Items from the Depression/Dejection Scale

#5 Unhappy

#32 Discouraged

#48 Helpless

Items from the Anger/Hostility Scale

#3 Angry

#24 Spiteful

#33 Resentful

APPENDIX D: Patient Diagnostic Form
Patient Diagnostic Information
(to be obtained from the medical chart)

Subject Number: _____ **Name:** _____

Clinic: Hayden Hahnemann Pulmonary MCP Pulmonary
Temple Head and Neck Temple Pulmonary

Doctor: **Date:**

Diagnosis:

COPD
_____ Emphysema
_____ Chronic bronchitis
_____ COPD

Head and Neck Cancer
_____ Oral cavity
_____ Paranasal sinus and nasal cavity
_____ Salivary glands
_____ Oropharynx
_____ Nasopharynx
_____ Hypopharynx
_____ Larynx
_____ Metastatic squamous cell carcinoma
_____ Other: _____

Date of Initial Diagnosis (Month/Year): _____

Disease Severity (Please check below):

COPD

FVC (current/most recent): _____(_____) DATE: / /

_____

FEV1 (current/most recent): _____(_____)

FEV1/FVC (current/most recent): _____

_____

FVC (at initial diagnosis/earliest in chart): _____(_____) DATE: / /

FEV1 (at initial diagnosis/earliest in chart) _____(_____)

Head and Neck Cancer

Stage (current) :

DATE: / /

Stage (at initial diagnosis): _____

DATE: / /

FEV1/FVC (at initial diagnosis/earliest in chart) _____

Treatment:

Current Medications (Please list):_____

_____

_____

Surgery (Date of most recent surgery M/DD/YY):_____

Radiation: Yes No Chemotherapy: Yes No

Other (Please list additional treatment):

_____

_____

Previous Treatment:

_____

_____

Other Medical Conditions:

_____

_____

APPENDIX E: Informed Consent

MCP Hahnemann University
Consent to Participate
In a Research Study

1. **Subject Name: _____**

2. **Title of Research:** Predictors of Smoking Behavior in People with Head and Neck Cancer and Chronic Obstructive Pulmonary Disease

3. **Purpose of Research:**

You are being asked to participate in a research study. The purpose of this study is to assess how various problem-solving styles are related to a person's psychological reactions to either head and neck cancer or chronic obstructive pulmonary disease, and their smoking behavior. It is expected that 150 individuals will be enrolled in the study.

4. **Procedures and Duration:**

You understand that you will be asked to do the following:

(a) To provide information about yourself, such as your age, race, and medical treatment. Your name will not be on any materials retained in the study and this personal information will only be known to the research team.

(b) To allow us, the research team, to communicate with your doctor regarding your medical diagnosis.

(c) To fill out several brief questionnaires asking how you typically solve problems in living, how you are currently feeling, about current problems that you have due to either cancer or COPD, and questions about your past and current smoking behaviors. **You understand that information about your smoking, both past and current, will NOT be revealed to your doctor.** The questionnaires are expected to take approximately 20-30 minutes, and you are asked to complete

them in the physician's office. You will be provided a private room in which to complete these questionnaires. You will be asked to complete these questionnaires only one time, during today's visit.

(d) You will be contacted in three months and asked to complete a telephone interview regarding your smoking status and smoking behavior since your participation in this study. You understand that the telephone interview is expected to take approximately 5 minutes.

5. **Risks and Discomforts:** You have been told that the risks and/or discomforts of being in this study include:

(a) You may worry about who may have personal information about me. *Since information about you is private, when the researchers talks about the results of this project, all information will be put together with everyone else's. Only the professionals involved in this study will see any of the answers.* **However, your physician and the care coordinator, who are a part of the research team, will not have access to your personal smoking history.**

(b) Filling out the questionnaires may increase your negative feelings by making you think about your current problems. *If these feelings or thoughts make you feel worse, you can discuss them with any member of the project. They will try to understand these feelings and be of help to you*.

6. **Benefits:** You understand the following benefits may occur as a result of your participation in this study:

(a) You understand that you may not directly experience any benefits.
(b) Your participation may help future people with head and neck cancer and COPD by helping researchers know more about the importance of problem-solving styles in coping with medical illness. This information can lead to the development of important educational and counseling programs.

7. **Reasons for Removal from Study:**

You may be required to stop the study before the end for any of the following reasons:

(a) If required by a change in medical condition;

(b) If all or part of the study is discontinued for any reason by the sponsor or government agencies; or

(c) If others in this study experience harmful reactions.

8. **Voluntary Participation**

You understand that being in this study is voluntary. Your health care will not be affected in any way if you decline to be in, or later withdraw from, this study.

9. **In Case of Injury**

You have been told that if you have any questions or believe you have been injured in any way by being in this research project, you should contact Dr. Christine M. Nezu at (215)762-3677. If you have an adverse reaction as a result of this study, you should contact the MCP Hahnemann University Research Administration Office at (215)762-3453.

10. **Consent to Use Research Results and Confidentiality of Records:**

All data obtained in this study will be kept confidential. In any publication or presentation of research results, your identity will be kept confidential, but there is a possibility that records which identify you may be inspected by authorized individuals and agencies such as the Food and Drug Administration, the Institutional review Board (IRB), or employees conducting peer review activities. I consent to such inspections and to the copying of excerpts from my records, if required by any of these representatives.

You medical records will be handled as are the records of all patients with similar medical condition entering MCP Hahnemann University Hospitals for clinical treatment.

11. **Other Considerations**

If new information becomes known that will affect you or might change your decision to be in this study, you will be informed by the investigator. If you have any questions at any time about this study or your rights as a research participant, I may contact the Office of Research Administration at (215)762-3453.

12. **Participant Certification:**

I hereby certify that I do not fit within any of the following categories:

- known history of mental illness diagnoses including bipolar disorder, psychosis, and/or mental retardation
- under 25 years of age or over 80 years of age

12. **Consent**

- I have been informed of the reasons for this study.
- I have had the study explained to me.
- I have had all of my questions answered.
- I have carefully read this consent form, have initialed each page, and have received a signed copy.
- I give consent voluntarily.

_____
Participant Name (please print)

_____ _____
Participant's Signature Date

_____ _____
Investigator/Individual Obtaining Consent * Date

_____ _____
Witness to Signature Date

List of Individuals Authorized to Obtain Consent*

*	Name	Telephone Number
	Kim P. Baron, Ph.D.	(215) 762-7625
	Christine M. Nezu, Ph.D.	(215) 762-3677

APPENDIX F: Informed Consent

Temple University Hospital
Consent to Participate
In a Research Study

Predictors of Smoking Behavior in People with Head and Neck Cancer and Chronic Obstructive Pulmonary Disease

Principle Investigator: Edward Volkman, M.D.

Co-Investigators: Kim Baron, M.A.

Daniel J. Kelley, M.D.

John Travaline, M.D.

Participant Name: _____ **ID#** _____

IRB Protocol # _____

PURPOSE OF THE STUDY

I am being asked to participate in a research study. The purpose of this study is to assess how various problem-solving styles are related to a person's psychological reactions to either head and neck cancer or chronic obstructive pulmonary disease, and their smoking behavior. It is expected that 150 individuals will be enrolled in the study.

DESCRIPTION OF THE PROJECT

I understand that I will be asked to do the following:

(a) to provide information about myself, such as my age, race, and medical treatment. My name will not be on any materials retained in the study and this personal information will only be known to the research team.

(b) to allow the research team to communicate with my doctor regarding my medical diagnosis.

(c) to fill out several brief questionnaires asking about how I typically solve problems in living, how I am currently feeling, about current problems I have due to either

cancer or COPD, and questions about my past and current smoking behaviors. **I understand that information about my smoking, both past and current, will NOT be revealed to my doctor**. The questionnaires are expected to take approximately 20 - 30 minutes, and I am asked to complete them in the physician's office. I will be provided with a private room in which to complete these questionnaires. I will be asked to complete these questionnaires only one time, during today's visit.

(d) I will be contacted in three months and asked to complete a telephone interview regarding my smoking status and smoking behavior since my participation in the study. I understand that the telephone interview is expected to take approximately 5 minutes.

CONFIDENTIALITY STATEMENT

All documents and information pertaining to this research study will be kept confidential in accordance with all applicable federal, state and local laws and regulations. I understand that medical records and data generated by the study may be reviewed by Temple University's Institutional Review Board, the study sponsor, and the United States Food and Drug Administration (FDA) to assure proper conduct of the study and compliance with federal regulations. I understand that the results of this study may be published. If any data are published, I will not be identified by name.

VOLUNTARY PARTICIPATION STATEMENT

I understand that my participation in this study is entirely voluntary, and that refusal to participate will involved no penalty or loss of benefits to me. I may discontinue my participation at any time without penalty or loss of benefits.

COMPENSATION STATEMENT

I understand that I will receive no compensation for this study.

INSTITUTIONAL CONTACT

If I have any questions about my rights as a research subject, I may contact the Institutional Review Board Manager, Ruth S. Smith at (215) 707-3249.

If I have any questions about research-related injuries, I may contact Dr. Edward Volkman at (215) 707 - 5343.

STANDARD INJURY STATEMENT

I understand that if I sustain an injury as a result of participation in this study, only physician's fees and medical expenses not covered by my medical and hospital coverage or other third party coverage will be paid at no cost to me. I understand that financial compensation for such injuries is not available. I understand that I have not waived any of the legal rights which I would otherwise have as a participant in an Investigational study.

COSTS STATEMENT

I understand that the tests required by the study will be provided at no cost to me.

TERMINATION STATEMENT

The investigator or the sponsor may terminate my participation in the study without my consent.

FINAL STATEMENT AND SIGNATURE

This study has been explained to me, I have read the consent form and I agree to participate. I have been given a copy of this consent form.

_____	_____
Participant	Date
_____	_____
Investigator / Co-Investigator	Date
_____	_____
Witness	Date

APPENDIX G: Three-Month Follow-Up

Predictors of Smoking Behavior in People with Head and Neck Cancer and Chronic Obstructive Pulmonary Disease

Telephone Interview
3 Month Follow-Up

I. General Information

Subject Number: _____

Name: _____

Date if Initial Participation: _____

Clinic: Hayden Hahn. Pulmonary MCP Pulmonary
Temple Head and Neck Temple Pulmonary Doctor: _____

Date of Follow-Up Interview: _____

Telephone Number: Home (_____) _____-_____
Work (_____)_____ - _____

Best time and place to contact you: Time: _____ Place: Home Work

Okay to leave a message? Yes No

Smoking Status at Initial Interview: Smoker Ex-smoker
(From Initial Contact, Record: Amount Smoked: _____ (or) Date Quit: _____)

II. Illness Variables

What is your current diagnosis? _____

What is your current treatment? _____

III. Smoking Variables **Remind participant about confidentiality.**

A. Have you smoked at least one puff of a cigarette in the past 7 days?

Yes No

Have you smoked at least one puff of a cigarette in the past 3 months (since your initial participation in the study? Yes No

If yes to either:

On average, how many cigarettes are you *currently* smoking per day? _____

Over the past three months, how many cigarettes, *on average*, did you smoke per day? _____

Over the past 3 months, when your smoking is the heaviest, how many cigarettes do you smoke per day? _____

Do you inhale? Always Sometimes Never

Over the past 3 months, when you did smoke, when do you smoke the heaviest (check one box only): Mornings Afternoons Evenings

Over the past 3 months, on the days that you did smoke, how soon after you wake/woke up do/did you smoke your first cigarette?

____ immediately

____ within 30 minutes

____ between 30 minutes and one hour

____ beyond one hour

Which cigarette is/would be/was the most difficult to give up?

First cigarette in the morning

After meals

During or after stressful situations

During social occasions

In what situations do you smoke?

In public

At work

At home

In the presence of certain relatives

In the presence of my children

At meetings

Inside the home of non-smokers

In my car when non-smokers are with me

In other peoples cars

In restaurants

Other. Please specify _____

B. Over the past 3 months, did you try to cut down or limit your smoking?

Yes No

If Yes:
How many times have you *attempted* to stop smoking? _____

During the last 3 months, how many times have you stopped smoking *for at least one day*?
_____

During the last three months, what is the longest time you had stopped smoking?

Less than a day
At least one day but less than a week
At lease one week but less than a month
At least one month but less than two months

At least two months but less than three months

What methods, if any, did you use to stop smoking (e.g., cold turkey; nicorette gum; nicotine patch; support group; Zyban, etc.)? _____
What symptoms did you experience when you stopped smoking? (Please check all that apply):

Craving	Anxiety	Restlessness
Decreased heart rate	Increased eating	Difficulty concentrating
Irritability	Other: _____	

PLEASE THANK THE PERSON FOR THEIR PARTICIPATION IN THE STUDY.

VITA

Kim Phillips Baron

Education

Drexel University, Ph.D., Clinical Psychology
Temple University Hospital, Pre-Doctoral Internship
Lehigh University, BA (1992)
Major: International Careers - Business track; Minor: French
The Horace Mann High School

Honors and Awards

Hedy K. Singer Award for "Excellence in Psychotherapy"
Fellow, Center for Mind/Body Studies
Graduate Travel Award, MCP Hahnemann University
Dean's List, Foreign Language Scholarship, and Phi Eta Sigma Honors Society, Lehigh University

Clinical, Research, and Professional Experience

Temple University Hospital, Philadelphia, PA, Pre-Doctoral Internship
MCP Hahnemann University Student Counseling Center, Philadelphia, PA
Center for Mind/Body Studies, Philadelphia, PA
Genesis Cancer Coping Project, Philadelphia, PA
Project S.T.O.P. (Sexual Treatment of Persons with Mental Retardation), Philadelphia, PA
Beth Israel Medical Center, New York, NY
The Mount Sinai Medical Center Therapeutic Nursery, New York, NY
Lenox Hill Hospital, New York, NY

PUBLICATIONS

Nezu, C.M., Tsang, S., Lombardo, E.R., and **Baron, K.P.** (2002). Alternative and complementary therapies in health psychology. In A.M. Nezu, C.M. Nezu, and P.A. Geller (Eds.). *Health Psychology* (Volume 9 of the 12-volume series, Comprehensive Handbook of Psychology). New York: Wiley.

Nezu, C.M., Nezu, A.M., **Baron, K.P.**, & Roessler, E. (2001). Alternative Therapies. In G. Fink (Ed.), *Encyclopedia of Stress*. New York: Academic Press.

Epstein, G., Barrett, E.A.M., Halper, J.P., Seriff, N., **Phillips, K.** & Lowenstein, S.

(1997). Alleviating asthma with mental imagery: A phenomenological approach. *Alternative & Complementary Therapies*, February: 42-52.